Women's orgasm

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CHAPTER 21

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CONTENTS

Women's Orgasm

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A. INTRODUCTION TO THE WOMEN'S ORGASM

I. DEFINITION OF WOMEN'S ORGASM

More than one author has commented on the extensive literature that exists about the human female orgasm. It has been discussed from clinical, ethological, philosophical, physiological, psychological, sociological and typological perspectives [1]. Symons [2, p. 86] observed that although "the human female orgasm definitely exists [it] inspires interest, debate, polemics, ideology, technical manuals and scientific and popular literature solely because it is so often absent !" It is clear that natural selection has not favored females who could orgasm easily, hence it is not likely an essential feature of the reproductive process. Even its definition is hard to pin down because enigmatically it has both nomothetic (the study or discovery of general laws) and idiographic (individual's performance) aspects. Because the exact neural activity of the cerebral neuronal discharge is so poorly understood, most definitions use reported or observed physical changes (usually muscular and cardiovascular) with an emphasis that this is the culmination or most intense moment of sexual arousal. Levin [3] tabled some 13 definitions by authors from a variety of backgrounds, and 20 years later Mah & Binik [4] repeated the exercise with a doubling of the author definitions. They divided them into three groups : those primarily with a biological perspective, those with a psychological one and those with an integrated biopsychological perspective. The authors still had to conclude that a satisfactory universal definition of orgasm could not be accomplished. Considering that the human orgasm is regarded as the ultimate state of ecstatic feeling without recourse to drugs, it is remarkable how few of the definitions incorporated the word "pleasurable". The more recent reports that women with complete spinal cord injury (SCI) can experience orgasm further complicate an all-encompassing definition of orgasm [5]. A major problem in defining orgasm is the emphasis that is given to the subjective or self-report as opposed to objective physiological signs. Despite all these difficulties, it is useful to devise at least an operational definition for women's orgasm, thus:

"An orgasm in the human female is a variable, transient peak sensation of intense pleasure, creating an altered state of consciousness, usually accompanied by involuntary, rhythmic contractions of the pelvic striated circumvaginal musculature, often with concomitant uterine and anal contractions and myotonia that resolves the sexually-induced vasocongestion (sometimes only partially), usually with an induction of well-being and contentment".

II. TYPOLOGIES OF WOMEN'S ORGASM

Typologies of orgasm intriguingly only exist for women ; those for men have not been explored even though some therapists have suggested that they exist [6]. Most of the typologies [7] are from self-reported perceptions of women distinguishing orgasmic sensations induced by clitoral stimulation (warm, ticklish, electrical, sharp) from those obtained by vaginal stimulation (throbbing, deep, soothing, comfortable). A frequently quoted typology is that of Singer [8, pp. 72-73], a philosopher with no experience of laboratory studies, but who analyzed the descriptions of orgasms from a limited literature and characterized three :

1. "vulval", rhythmic contractions of the vagina activated by either clitoral or coital stimulation,

2. “uterine”, no vaginal contractions but accompanied by apnoea and gasping activated during coitus alone and largely due to penile-cervix contact, and
3. “blended”, containing elements of both vulval and uterine orgasms activated by coitus and accompanied by apnoea.

Great play was made about the importance of apnoea and especially cervical stimulation. The latter was not so much about stimulation of the cervix per se, but rather its displacement by the thrusting penis causing the uterus to rub against the peritoneum claimed by Singer & Singer [9] to be a “highly sensitive organ”. Unfortunately, the total evidence for this typology rests on limited scientific observations obtained in remarkably few individuals [10]. Furthermore, the role of the cervix in a woman's sexual response (viz uterine orgasm) is unclear. One reviewer declared that, depending on the studies cited, any position can be supported [11], while a more recent reviewer concluded that evidence for and against a role of the cervix in orgasm is weak and that observational studies cannot answer the question [12]. Another difficulty with this typology is that according to Ingelman-Sundberg [13], the anterior vaginal wall acts like a hammock around the urethra, and during coitus the penis stretches two of its ligaments that insert around the base of the clitoris thus effectively stimulating it during thrusting. If this mechanism operates in all penile-vaginal coital penetrations it would create both “uterine” and “vulval” stimuli or the so-called “blended” stimulus.

Although Masters & Johnson [14] claimed that all orgasms in females were physiologically identical regardless of the source of stimulation, they did not have the instrumentation to obtain detailed muscular recordings for possible differences between clitoral and so-called G-spot (anterior vaginal wall) induced orgasms. There is now some limited physiological laboratory evidence that different patterns of uterine (smooth muscle) and striated pelvic muscular activity may occur with vaginal anterior wall (G-spot ?) stimulation as opposed to clitoral stimulation; one such set of recordings is shown in Levin [10]. The case for such a dual typology may well be made more credible by this type of evidence.

Bohlen, Held, Sanderson and Ahlgren [15] characterized the vaginal muscular contractions at orgasm in their 11 nulliparous subjects into three typologies which varied greatly in terms of orgasm duration. The typologies were: those that had regular rhythmic contractions (mean duration of orgasm 13 seconds), those that had regular contractions with later irregular ones (mean orgasm duration 50.6 seconds), and those that had no regular rhythmic contractions during their orgasms (mean duration of orgasm 24.4 seconds). To create a muscular typology of such a range with so few subjects is premature but unfortunately no further studies have been reported. There is a need for simultaneous recordings of the uterine and vaginal motility at orgasm in a large sample of women.

III. GENDER DIFFERENCES IN ORGASM

Written descriptions/accounts of orgasms by men and women with any obvious gender clues removed could not be differentiated by sex when read by other men and women [16]. This suggests that men and women share common mental experiences during orgasm. Four differences in male and female orgasms, however, have been proposed :

1. unlike the male, the female can have repeated (multiple) orgasms separated by very short intervals [14, p. 131]
2. the female can have an extended orgasm lasting for a long time (so-called “status orgasmus”, [14, p. 131]),
3. there are differences in the recorded pattern of pelvic muscular contractions ; specifically, men have a divided rhythmic pattern not seen in women [15], and
4. once the male orgasm is initiated, its further expression is automatic even if sexual stimulation is stopped ; if stimulation is stopped in the middle of either clitoral-induced or vaginal-induced female orgasm, the female orgasm is halted [17, p. 121].

IV. WHY DO WOMEN HAVE ORGASM ?

It is generally accepted that female orgasms are not essential for reproduction. Any benefit for various aspects of female biology is, as of yet, unclear. There are a number of explanations from the literature regarding why the human female has orgasms :

1. the reward of intense pleasure for acceptance of the danger of coitus with its possibility of pregnancy (and of possible death in childbirth)

2. to end coitus

3. for resolving pelvic vasocongestion/arousal

4. for resolving vaginal tenting (allows the cervix to enter the seminal pool)

5. orgasmic uterine contractions may create a possible sperm upsuck

6. to create arousal in the male by felt vaginal contractions on the penis and cause ejaculation (capturing the semen)

7. for inducing lassitude to keep the female horizontal and thereby reducing seminal "flowback"

8. because of the difficulty in attaining orgasm (especially coitus), orgasm acts as a "Mr. Right" indicator and aids the creation of a strong "pair bond"

9. to create the loss of body boundaries and separateness allowing a merger or fusion with the chosen coital partner

10. to create psychological resuscitation-like an electric shock redistributing the potentials of the brain

11. to release oxytocin which affects motility of the uterus and fallopian tubes and possibly to induce bonding feelings and emotions

12. to release ADH for the possible contraction of uterine musculature and to inhibit urination and delay sperm losses from "flowback"

13. for manipulation of the uptake or rejection (flowback) of deposited sperm

14. by its activation of muscular contractions and the concomitant increased blood flow, orgasms maintain the functionality of the genital tract [18].

The history of the claimed importance of the female orgasm to reproduction is full of speculative functions with little or no scientific data for their support. Orgasmic coitus was said to activate ovulation and close off the womb to air, thus facilitating conception [19]. When it was later shown that the human female was a spontaneous ovulator at mid-cycle unconnected with coitus, the discourse re-focused on the role of uterine orgasmic contractions in the movement of ejaculated spermatozoa through the cervix into the uterus and then fallopian tubes. Singer [8], in the light of his protagonism for his dual typology of female orgasm (so-called uterine and vulval), published an extensive discussion about fertility and the female orgasm which explored issues of

1. uterine suction,

2. extrauterine factors affecting the mechanics of uterine suction (viz vaginal tenting), and

3. the ejaculatory timing in coitus (i.e., ejaculation must occur before orgasm to assist sperm transport, but see discussion below, re Baker & Bellis's [20] proposals).

It has often been suggested that uterine suction created by the contractions of the uterus at orgasm would facilitate the transport of spermatozoa into the uterus and then to the fallopian tubes. Evidence now shows the fastest transport of spermatozoa into the human uterus is actually in the sexually unstimulated condition [21-22]. One essential feature of sexual arousal of the female genitalia is to create the expansion of the vagina (vaginal tenting) and elevation of the uterocervix from the posterior vaginal wall to reduce the possibility of the rapid entry of ejaculated spermatozoa into the uterus. This gives time for the initiation of the decoagulation of the semen and the capacitation of the spermatozoa to begin, and it reduces the chance of incompetent sperm being too rapidly transported into the fallopian tubes. The female orgasm, by dissipating arousal and initiating the resolution of the tenting, will in fact allow the earlier entry of the spermatozoa into the cervical canal and their subsequent rapid transport to the fallopian tubes, although there is some evidence that the motility of the uterus is reduced after sexual arousal and orgasm [see 21]. Not experiencing orgasm during coitus may thus have a reproductive utility unrealized by previous authors.

Another suggested reproductive function of female orgasms, initiated either from coitus or masturbation, is their use by women to manipulate the ejaculate in the vagina [20, 23]. This highly contentious claim is based on the amount of "flowback" (semen/fluid) lost from the vagina. According to these authors, the amount of flowback containing spermatozoa varies exquisitely with the precise timing of the female orgasm in relation to the time of deposition of the ejaculate into the vagina. Low sperm retention was said to be associated with female orgasms earlier than one minute before vaginal deposition while maximum retention was claimed with orgasms from greater than 0 to 1 minute after deposition. If orgasm occurred earlier than 1 minute before the ejaculate, deposition sperm retention was the same as when there was no orgasm. According to Baker and Bellis [20] the effect of orgasm on sperm retention lasts only for the period of one minute before semen deposition and up to 45 minutes later. During the period when the seminal fluid was coagulated (15 minutes),

orgasms have a significantly reduced effect on sperm retention. These complicated scenarios for the effects of female orgasm on sperm retention still depend on a contractile uterine "upsuck" sperm mechanism. During orgasm, the cervix "searches and dips" into the seminal pool, and the orgasm-induced movements either facilitate the dipping, and/or the mixing of the cervical mucus with the pool and/or increase the time that the cervix is in the pool.

The coital scenario previously described [18, 21] where "vaginal tenting" removes the cervix from the seminal pool, thus delaying the uptake of sperm in order to allow the initiation of its liquefaction and the capacitation of the sperm, contrasts dramatically with the above hypothesis of Baker and Bellis. The crucial factor is the presence or absence of vaginal tenting. Masters & Johnson [14, pp. 113-114] reported it is only absent in women with retroverted or retroflexed positioned uteri but Singer [8] speculated that it may not occur in either his "uterine" or "blended" orgasm. The only evidence for this speculation is a preliminary report by Perry & Whipple [24] who claimed that "tenting never occurred in response to Grafenberg (G-spot) stimulation". It led to a "direct descent of the uterus and places the cervix immediately and directly into the seminal pool, where it may facilitate conception". So-called "uterine" orgasms in Perry & Whipple's preliminary analysis would facilitate conception while "vulval" ones would not. The cervical disposition created by the former orgasm would allow rapid sperm entry before capacitation had been initiated and thus facilitate the uterine/tubal entry of sperm incompetent to fertilize. Ultrasound imaging of face-to-face coitus did not show penile cervical contact [25] as would be expected if Singer's "uterine" orgasm were to be induced while the recent imaging by MRI of the relationships between the penis, cervix and the uterus during coitus and masturbation has confirmed the concept of vaginal tenting in a relatively small number of couples [26-28]. It is thus likely that tenting occurs in face-to-face coitus and that its effect on fertilization is positive [18, 21]. It is clear that more MRI scans of human coital activity are needed to confirm this definitively.

A less controversial claim of one of the functions of orgasm to aid in the reproductive process is that if the female allows the expression of orgasm during coitus, its contractions of the vagina can excite the male ejaculate thus allowing the female to capture the sperm of her chosen inseminator. Orgasm increases the secretion of prolactin. If this increased secreted prolactin in plasma is able to enter into the vaginal, cervical or uterine fluids, it might be a factor in influencing the entry of calcium into the sperm as it acts as a physiological ionophore. This action could play a role in the activation of spermatozoa in the female tract [29]. Finally, one area of the putative involvement of orgasm in reproduction that is generally not discussed is its use to induce and encourage the first stage of or to relieve the pain of childbirth [30]. Some women have spontaneous orgasms during the passage of the fetal head through the vaginal canal, probably through the stretching activation of the cluster of erotic sites along the anterior vaginal wall.

B. OBJECTIVE SIGNS OF ORGASM

Orgasm is a subjective experience accompanied by a number of physiological body changes. The degree to which these changes vary between individuals is not known. Males have little difficulty in identifying orgasm because although orgasm and ejaculation are created by distinct mechanisms (see [22] for references), it is rare for the former not to accompany the latter. In women, the achievement of orgasm appears to be less facile than for males and recognizing that it has occurred can be difficult for some. Thus, just asking previously anorgasmic subjects whether or not they experienced an orgasm after a treatment or therapeutic session is somewhat unreliable. An objective indicator(s) that an orgasm has occurred to confirm or inform any subjective report would be of real clinical and therapeutic value.

Objective indicators of orgasm have been sought after for many years often with little regard for their utility in the clinical context. Kinsey, Pomeroy, Martin & Gebhard [31] proposed "the abrupt cessation of the ofttimes strenuous movements and extreme tensions of the previous sexual activity and the peace of the resulting state" as the most obvious evidence that orgasm had occurred and of identifying it in the human female. Masters & Johnson [14] described the onset of orgasm as a "sensation of suspension or stoppage". Clearly, however, the indicator must involve a body change that is unique to orgasm, which rules out simple measures like peaks of blood pressure, heart and respiratory rates or even a naïve subject's own vocalizations indicating it is impending or is occurring because such peaks can arise

even if no orgasm occurs. Remarkably, most of the so-called objective indicators of female orgasm rely on the original, nearly 40-year old observations and descriptions of Masters & Johnson [14]. They are of three types : prospective- those indicating an impending orgasm, current- those occurring during the actual orgasm, and retrospective- those indicating that an orgasm has occurred : they are listed in **Table 1** and described in detail below. Surprisingly, even such a simple classification system has its problems as it is possible to place some of the indicators in either the current or retrospective category depending on the chosen definition of the initiation of the orgasm. It is unclear whether orgasm should be defined as starting when the woman first mentally perceives it, or whether it starts when the first physical manifestation occurs. Kinsey, Pomeroy, Martin & Gebhard [31] tried to limit the definition of orgasm to the sudden and abrupt reduction of sexual tension. The "spasms" into which individuals are thrown was argued to be the "after effects" of the orgasm while the "vaginal spasms" were regarded simply as "extensions of the spasms that may involve the whole body after orgasms". Hite [32] also regarded orgasm as a brief intense feeling followed by contractions.

I. PROSPECTIVE CHANGES - LABIA MINORA COLOR CHANGES

The paired labia minora on either side of the vaginal introitus are continuous ventrally with the prepuce and frenulum of the clitoris and join the labia majora posteriorly. They are composed of adipose tissue, connective tissue rich in elastic fibers with smooth muscle fibers and numerous wide veins. The amount of cavernous tissue present is variable in individuals, in some it is extensive and in others it is hardly present. The tissue is a spongy mass like that of the clitoris except that it does not have a capsule around it and has fewer nerves in the trabeculae. Merkel tactile discs and genital corpuscle (Dogiel/Krause) are found in the prepuce and ventral part with a rich network of nerves. Free nerve endings (pain ?) lie just beneath the germinative stratum and Pacinian corpuscles (pressure ?), and are frequently noted along the courses of nerves [33-34]. Numerous eccrine sweat glands and a few apocrine glands are present.

During sexual arousal, the labia become engorged with blood and increase in size adding on about 1 cm to the length of the vagina. According to Masters &

Table 1. Characteristic changes that occur during orgasm (mainly from Masters & Johnson, 1966).

Extragenital	
Respiration	- usually peaks to a maximum at initiation of orgasm (>40/min)
Blood pressure	- usually peaks at initiation of orgasm and then decreases (systolic +30-80mmHg, diastolic + 20-40mmHg)
Heart rate	- usually peaks at initiation of orgasm then decreases (110-180 beats/min)
Sex flush	- superficial maculo-papular (vasocongestive) over epigastrium, anterior chest wall spreading to neck and face has greatest intensity at orgasm
Perspiration	- approximately 33% women in back, thighs and chest
Breasts	- No specific change
Nipples	- No specific change
Areolae	- rapid detumescence of congestion leading to transient corrugation
Myotonia	- contractions in neck, face, arms & legs may become spastic, carpopedal spasm (generalized)
Genital/Pelvic	
Clitoris	- No specific change
Labia (majora and minora)	- No specific change
Vagina contractions	- rhythmic contractions (throbbing) of outer third (orgasmic platform) due to pelvic striated muscle
Uterus	- expulsive contractions of smooth muscle
Rectal sphincter	- contractions mirroring vaginal contractions
Cervix	- dilation of os immediately after orgasm lasting 20-30 minutes
Hormonal	
Anterior pituitary	- increased secretion of prolactin
Posterior pituitary	- increased secretion of ADH (Vasopressin) and Oxytocin
VIP	- increase in plasma VIP

Johnson [14, pp. 41-42], once their initial engorgement has been induced, vivid color changes occur with further sexual arousal. The color changes were said to be "clinically pathognomic" of impending orgasm as the claim was made that "No premenopausal woman has been observed to reach plateau-phase levels of sexual tension, develop the 'sex skin' color changes and then not experience an orgasm". After the orgasm occurs, the color changes rapidly within 10-15 seconds, from deep red to light pink. If the color change takes place and then the sexual stimulus is removed, it rapidly fades well ahead of the slower loss of the engorgement. No other study has confirmed these findings despite this highly specific claim of impending orgasm on the minora labia color change. In fact, there has been little detailed study of the minora labia apart from the suggested mechanism by which they become lubricated [35] and that their increased temperature during sexual arousal has been used as an objective indicator of arousal [36] prior to and after orgasm [37]. The color changes of the labia are presumably due to the changing hemodynamics of the tissue in relation to increased blood flow, tissue congestion and tissue metabolism (oxygen consumption) indicating the balance between oxygenated (red/pink) and deoxygenated or reduced hemoglobin (blue). The blue color of cyanosed mucous membranes occurs when the absolute amount of reduced hemoglobin is greater than 5 gms/100 mls blood. The percentage saturation oxygenation of the blood (s02) is usually measured by light absorbancy but no quantitative studies have been made on the labia minora during sexual arousal. In the basal state the vaginal surface which has a very low p02, practically hypoxic [38], rapidly increases during sexual arousal up to a maximum at orgasm. Repetition of this study [39] confirmed the vaginal findings and showed that the labia minora followed a similar pattern.

II. CURRENT INDICATORS OF ORGASM

1. Vaginal rhythmic contractions

The resting vagina is a collapsed tube lined with a stratified squamous epithelium, approximating an elongated S-shape in longitudinal section and an H-shape in cross-section, invested with an outer longitudinal and inner circular layer of smooth muscle. It is anchored amid a bed of powerful, voluntary, striated muscles (pelvic diaphragm, consisting of the pubococcygeus and iliococcygeus muscle) of which the pubococcygeus has fibers that insert into the smooth muscle [22, 40]. Balloon recordings of the pressure inside the vagina show that just before orgasm is initiated there is a slow increase, probably due to an increase in tone of these circumvaginal muscles ***(Figure 1)***, although the tone of the vaginal smooth muscle per se may also be involved. According to Masters & Johnson [14, p. 118], the contractions recorded in the vagina begin some 2-4 seconds after the subjective appreciation of the start of the orgasm. They occur in many pre- and postmenopausal women and are due to the activation of the circumvaginal striated muscles (especially the pelvic diaphragm, bulbospongiosus, ischiocavernosus) which involuntarily contract in 0.8 second repetitions. This squeezes the outer third of the vagina (designated the "orgasmic platform" by [14]) with some force that gradually becomes weaker as the interval between contractions increases.

Contractions were not thought to be the primary initiator of the orgasmic experience because they began a few seconds after the woman perceived that orgasm had started (but see later section on "What triggers female orgasm ?"). Their number (and power) varies enormously between individuals and is obviously dependent on the duration of the orgasm and the strength of the pelvic musculature. Masters & Johnson reported that the stronger the orgasm the greater the number of contractions and, thus, indirectly the longer the duration of orgasm (as each contraction was approximately 0.8 seconds apart). However, if the number of contractions and their approximate duration are multiplied together this gives an approximate duration of each grade of orgasm ; "mild orgasms" had an average of 3 - 5 contractions (2.4 to 4 seconds long), "normal" ones 5 - 8 (4 to 6.4 seconds long) while "intense" orgasms had 8 - 12 contractions (4 to 9.6 seconds long). This claim and quantification was given without any supporting data. Using physiological (pressure) recordings, there has been difficulty proving any link between the contractions and the perceived intensity of the orgasm [41-42]. The durations of the orgasms recorded were mean 35.6 seconds (SD 24.5, n= 11) [15] ; mean = 19.9 seconds (SD= 12, n = 26) [43] ; mean = 21.9 seconds (SE 6.4, n=9) [42]). Bohlen, Held & Sanderson [41] reported, in their small group of subjects, a precise correspondence between the start of orgasm and the onset of regular "vaginal" contractions, the end of orgasms and the end of regular contractions was not observed. In some subjects,

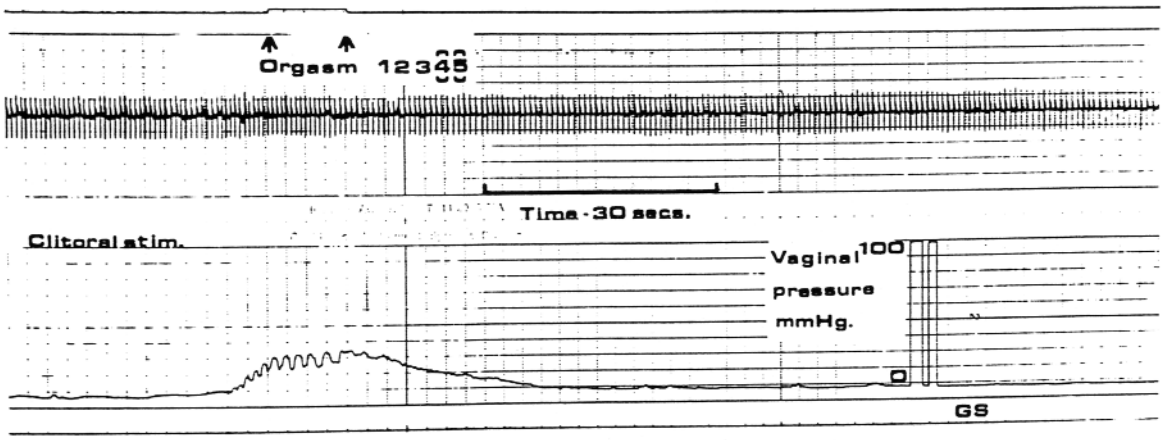

Figure 1 : Recording of changes in vaginal luminal pressure measured by a water-filled balloon (diameter 1.5 cm, length 3.5 cm) during self-induced orgasm by clitoral stimulation. The rise in muscular tone followed by 10-11 individual clonic contractions is clearly seen. The upper trace is the ECG recorded by telemetry. The start and finish of the orgasm (from the subject's reports) is shown by the two arrows while the subjective grading of the orgasm (on a scale of 1= poor to 5 = excellent) is given as 4-5.

the perceived start of orgasm preceded regular contractions by some 2-4 seconds (see report of [14] above), in others it coincided with the contractions, and a further group perceived orgasm followed the onset of contractions. Some of this variation could well depend on how accurate and quickly different subjects can report on their internal states. Contractions of the pelvic muscles at orgasm can also be monitored by recording their electromyogram that was undertaken in the study of Gillan & Brindley [44] using suction or fine wire electrodes in the circumvaginal muscles.

Masters & Johnson [14] confidently proposed that the vaginal contractions would "remove any doubt as to whether a women is pretending or experiencing orgasm", but other authors have noted that not all women who claim to experience orgasms show these contractions [3, 15, 41, 45-48]. It is especially interesting to note in this context that Bohlen et al. [15] stated that although two of their 11 subjects did not show distinct muscular evidence of orgasm they were not prepared to conclude that "physiological characteristics are more valid than self-reported perceptions for identifying orgasm" … "At least until more data are collected … we will continue our analyses of physiological changes based on subject's self-defined orgasms".

Unfortunately there has been no further detailed analysis of female orgasms so there is no large body of subjects who have had their vaginal muscular activity recorded during orgasm to assess the usefulness of the original contractile pattern classification. There has been little or no advance in the area since these studies in the 80's. The vaginal contractions have been used to objectively track the attainment of orgasmic capacity by a single initially anorgasmic subject [49].

It should also be noted that in the above account of the muscular activity at orgasm, there is no mention of the pattern of activity of the involuntary, longitudinal and circular vaginal smooth muscle during sexual arousal and at orgasm. Indeed, it is not even known whether they are relaxed, contracted or have a high tonus. One author has interpreted intravaginal balloon recordings from one subject as evidence for an enhanced tonus of the vaginal smooth muscle at arousal [50]. The slow rise in pressure in the vaginal lumen shown in Figure 1 may be due to such an increase in smooth muscle tone rather than that of the circumvaginal striated muscles; nevertheless, no routine, simultaneous recordings have been published with any instrumentation that has definitively separated their contractile activity in a series of subjects.

2. Uterine contractions

In their review on the "after-effects of orgasm", Kinsey et al. [31] commented that studies had shown that the "upper end of the uterus goes into rhythmic contractions of considerable frequency whenever there is sexual arousal". Masters & Johnson [14] however claimed, "Specific uterine patterns do not develop unless the individual study subject undergoes an orgasmic experience that is recognizable both by trained observers and by the individual

involved" (pp.116-119). Uterine motility was one of the physiological measurements that Masters & Johnson attempted, monitored by "intrauterine and abdominal electrode placement" (p. 116). Unfortunately, apart from this phrase, no details of the technique were ever published so we have no idea of the exact placement of the electrodes, their type or the equipment to which they were attached. The one published orgasmic record from the "intrauterine electrodes" (p. 117, Fig 8-2) is difficult to interpret, as it looks more like an increase in tone of the uterus rather than a series of contractions. Masters & Johnson claimed that the degree of contraction of the uterus paralleled "the study subject's subjective and the observer's objective evaluations of the physical and emotional intensity of the orgasmic experience". Few other investigators have examined uterine contractile function at orgasm. Fox, Wolff & Baker [51] used an intrauterine pressure transducer in a single subject who had sequentially a non-terminative and then terminative orgasm in the same sexual scenario, and uterine (and vaginal) contractions were recorded during the final orgasm. Caution is warranted in interpreting these conclusions because of the idiosyncratic orgasmic behavior of the female subject and the possible artifacts created by the large size and malfunctioning of the intrauterine transducer [1]. It has been proposed that orgasmic uterine contractions are the terminative signal for sexual arousal in multiorgasmic women [52] but again caution has been expressed [10]. Too few investigations have assessed orgasmic uterine contractions to make such a definitive statement.

3. Contractions of the anal sphincter

While voluntary contractions of the anal sphincter can occur during sexual arousal and are sometimes used by women to facilitate or enhance arousal, involuntary contractions of the anus occur only during orgasm [14, p. 34]. However, few measurements of anal contractions have been published aside from Bohlen and colleagues, who created a special anal pressure-measuring device [53] that could record the tone and contractions of the anal sphincter in both sexes during orgasm [15]. They reported that the anal contractions were synchronized with the vaginal contractions, yet the waveforms differed (vaginal = square wave, anal = sinusoidal), and those of the anus showed greater variability. Despite the obvious utility of recording the same muscular activity in both men and women at orgasm, little or no use has been made of the device since its creation in the 80's [15]. The genital and extra-genital changes that occur at or immediately after orgasm are listed in **Table 2**.

Table 2. Extragenital and genital/pelvic changes during female sexual arousal (mainly from Masters and Johnson, 1966);

Extragenital	
Hyperventilation	- from a basal of 14 breaths/minute to a max of 40
Tachycardia	- from a basal of 80 beats/minute to a max of 180
Hypertension	- Diastolic blood pressure elevated by 20-80 mmHg, Systolic blood pressure elevated by 80 - 100mmHg
Sex flush	- superficial maculo-papular (vasocongestive) rash initially over epigastrium and anterior chest wall then on neck, face and forehead
	Breast engorgement (increase in size)
	Areolae engorgement (increase in size)
Nipple erection	- elongation + 0.5 -1cm, base diameter + 0.25 - 0.5 cm
Myotonia	- elevated tension in muscles (legs, arms, neck, face (grimacing), abdomen)
Emission sounds	- sighs, moans, groans, grunts
Pupil dilation	
Genital/Pelvic	
Labia majora	- thins out and flattens against perineum
Labia minora	- expands in diameter, color changes, lubrication
Clitoris	- tumescence of glans (50% subjects) and shaft, retraction under clitoral hood
Vagina	- increases in blood flow, surface pO2, lubrication, Na+ and Cl-, length (+2.5 cm), tenting concomitant with utero-cervical elevation (width +3.75 to 4.25cm)
Uterus	- elevation into false pelvis (increase in size?)
Cervix	- no specific response

III. RETROSPECTIVE CHANGES

1. Areolae congestion and decongestion

The primary areolae, the usually large pigmented skin area around the nipple of the breasts, contain the follicles of Montgomery (small sebaceous glandular structures), occasional hair follicles, an underlying network of smooth muscle of interlacing bundles some 2 mm thick, blood vessels, elastic tissue, melanoblasts, Pacinian corpuscles, nerve fibers and a nerve (sympathetic ?) plexus [54-56]. The neural basis for areolar sensation was reported by Jones & Turner [57] to be "typically protopathic or thalamic in its character. The appreciation of the stimulus of cotton wool or testing hairs ceases abruptly at the margin of the specialised pigmented areola area". A later study [58] testing the cutaneous sensitivity of the breasts using Semmes-Weinstein monofilaments to obtain normal values found that the skin of the superior quadrant was the most sensitive, the areola less sensitive and the nipple the least sensitive. All areas were less sensitive the larger the breasts.

Swelling of areolae with arousal is likely due to both vasocongeston and smooth muscle contraction. The volume expansion can become so marked that the swollen/contracted areolae hide a large part of the base of the erect nipples making it look like they lose their erection. At orgasm, the loss of volume is so rapid that the areolae become corrugated before becoming flatter. This provides a "visual identification of the female orgastic experience" [14]. In the absence of an orgasm, the areolar detumescence is much slower and the corrugation does not develop. There is minimal study of areolar changes as an indicator that orgasm has taken place. Detailed descriptions of pre- and post-orgasmic changes of the areolae have not been fully explored, likely because they are difficult to monitor either quantitatively or qualitatively.

2. Enhanced post-orgasmic vaginal pulse amplitude (VPA)

Recordings of changes in the blood supply to the human vagina during sexual arousal to orgasm were made using the photoplethysmographic technique of Palti & Bercovici [59] with a superior vaginal photoplethysmograph created by Sintchak & Geer [60]. Geer & Quartararo [61] were the first to publish actual records of the vaginal pulse amplitudes (VPA) of the AC trace of each individual heart beat from their luminal, free-dwelling, vaginal photoplethysmograph before, at, and after orgasm in seven young women. Sexual arousal by masturbation caused an increase in the VPA signal compared to the basal values in all subjects, but immediately after the end of orgasm VPA was actually significantly greater than before orgasm in 5 of the 7 women (71%) and was not significantly less in the other two. The post-orgasmic period of maximum amplitude lasts for approximately 10-30 seconds and then VPA slowly returns to its basal value ***(Figure 2)***. This behavior has been observed in VPA signals recorded in other photoplethysmographic studies of orgasm [37, 44, 62-63]. There have been over 50 papers published using photoplethysmographic recording in the vagina but, unfortunately, these studies never took the induced sexual arousal to orgasm and beyond. Because there are so few photoplethysmographic recordings of the actual pre and post-orgasmic VPA traces published (less than a dozen), its utility as an indicator of orgasm in women is unclear. One basic

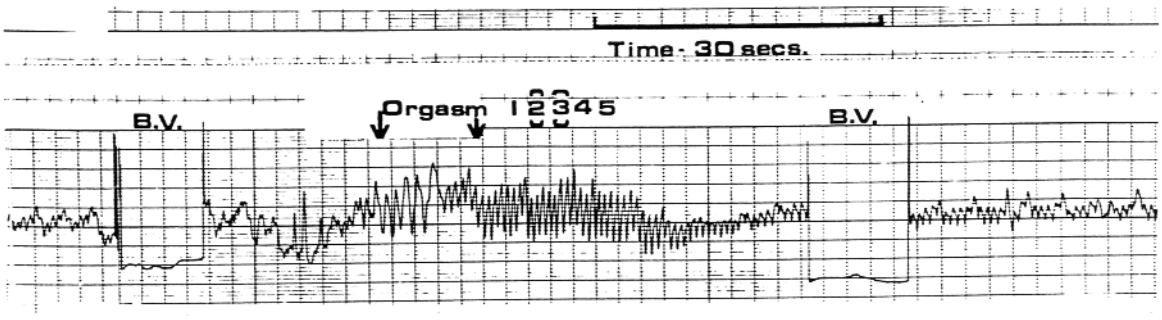

Figure 2 : Recordings of vaginal pulse amplitude (VPA) and vaginal blood volume (BV) obtained by a free dwelling luminal photoplethsymograph for i) basal, non- sexually stimulated trace, ii) BV (10 secs) during clitoral stimulation, iii) VPA during clitoral stimulation to orgasm and after, iv) BV (10 secs) post orgasm, iv) VPA post orgasm. The start and finish of the orgasm as reported by the subject is shown by the two arrows and is self-graded 2-3 on a scale 1= poor to 5 = excellent. The transient, enhanced, immediate post orgasmic VPA signal is clearly seen.

disadvantage in photoplethysmographic recording during orgasm using luminal free-dwelling instruments is that in many subjects the vaginal motility and orgasmic contractions can create severe movement artifacts in the records and make interpretation of the VPA signals extremely difficult. However, the development and exploitation of a suction photoplethsymograph [3] that is attached to the vaginal wall and thus moves with the wall would overcome such difficulties and allow interpretable recordings to be taken throughout orgasm. Contemporary collection and computerized processing of the VPA data also uses relatively long time periods (1 - 3 minutes) during an experiment. Given the effect is transient and usually only lasts for up to 30 seconds, the response can get lost when calculating overall mean amplitudes. Visual inspection of the data is essential.

3. Raised prolactin plasma levels after orgasm

Prolactin is a hormone secreted by the lactotrophic cells of the anterior pituitary gland. Its sequence of 199 amino acids was identified in 1969 [64] and was primarily thought to regulate lactation as early as the 1920's. Since its sequencing, prolactin has been shown to be involved in a huge variety of actions involving over 300 functions and is known to be produced or stored in a variety of cells. Its release from the pituitary lactrotrophs is unusual in that it is under tonic inhibitory control by the hypothalamus primarily via the neurotransmitter dopamine, although other substances and hormones can inhibit (e.g., Somatostatin, Endothelin, Acetylcholine) or facilitate (e.g., VIP, Oxytocin, TRH) its release [65]. Prolactin acts on the hypothalamic dopamine neurons to create a negative feedback loop to control its own release, similar to the other anterior pituitary hormones. The receptors for prolactin action are distributed in a variety of tissues (e.g., skin, bone, liver, male and female reproductive organs), but those of essential relevance are in the central nervous system (hippocampus, cortex, amygdala, hypothalamus) in areas known to regulate sexual behavior. A central role for prolactin in modulating sexual behavior and function in animals and in humans is now accepted.

Studies by Exton and colleagues have reported that prolactin secretion is not activated by sexual arousal per se but is specifically activated and doubled in plasma concentration by orgasm, whether generated by masturbation or coitus. This elevation occurs directly after orgasm and is maintained for approximately 60 minutes. Apart from its claimed indicator function for orgasm, the authors proposed that it also acts as a feedback control of sexual drive probably inducing its inhibition (refractory period), especially noted in the male after ejaculation and orgasm [65]. Females, of course, do not appear to have this refractory period after only one orgasm and can often undergo a whole series before satiation occurs [14]. If prolactin is the orgasmic-linked “off” switch for sexual arousal in men, why does it not act similarly in women ? Less well known, prolactin can also be released by tactile stimulation of the nipples in both women and in men especially when they are sexually aroused [66-67]. Thus, increase in prolactin secretion may be a retrospective signal that orgasm has indeed taken place. Its great disadvantage is the intrusiveness of its measure-it needs the pre-insertion of a butterfly cannula into a vein so that repeated venous samples can be withdrawn. Further studies need to be undertaken to see whether prolactin concentrations in other more easily accessible body fluids such as saliva, vaginal or cervical fluid or urine could be used **(Table 3).**

Table 3 . Objective indicators of female orgasm.

Prospective (indicating impending orgasm)
i) labia minora color changes
Current (occurring during actual orgasm)
ii) vaginal rhythmic contractions
iii) uterine contractions
iv) contractions of anal sphincter
Retrospective - (indicating an orgasm has occurred)
v) areolae congestion and decongestion
vi) enhanced post-orgasmic photoplethysmographic VPA
vii) raised prolactin plasma levels

4. The female prostate, female ejaculation, and the G-spot

In all the areas related to female sexuality, perhaps none have been surrounded with more aura than the concept of the G-spot [47] (named after Grafenberg who reportedly first anecdotally described the phenomenon [68]). Unfortunately, the popularization of this concept appears to have clouded our ability to make an accurate determination of its existence. To follow is a critical discussion of the scientific evidence for the female prostate, female ejaculation, and the G-spot.

Anatomic evidence from multiple autopsy studies

has demonstrated the presence of paraurethral glands [69-72]. In addition to the presence of these glands, histochemical evidence of prostatic acid phosphatase has been documented [70-71]. These reports provide strong evidence for the existence of periurethral glands in the female and for the presence of prostatic acid phosphatase. They also lead to the question of female ejaculation, and whether women expel fluid from their urethra concomitant with orgasm from G-spot stimulation, and what the components of this fluid are.

After the popularization of the term G-spot, a number of questionnaire studies reported that a significant number of women have acknowledged that they expelled fluid through their urethra at the time of orgasm [73-75] but these reports were anecdotal and did not provide any evidence of the source of fluid. Additionally, the reports were often preceded by the women hearing or being educated about the topic before they gave report of their own situations. This may have provided a suggestion to women that the correct answer was to say that they did ejaculate. There is essentially no scientific evidence to support the belief that women ejaculate with a fluid distinguishable from urine at the time of orgasm [77]. Laboratory studies [68, 77-79] have not revealed consistent evidence for any anatomical structure or "spot" on the anterior vaginal wall apart form the known paraurethral glands and spongiosal tissue around the urethra which could create sexually pleasurable sensations when stimulated.

5. What triggers women's orgasm ?

Women's orgasms can be induced by erotic stimulation of a variety of genital and non-genital sites. The clitoris and vagina (especially the anterior wall including Halban's fascia and urethra) are the most usual sites of stimulation, but stimulation of the periurethral glans [10], breast/nipple or mons ([14, p. 54; 66]), mental-imagery or fantasy [14, 80] or hypnosis [1] have also been reported to induce orgasm. Orgasms have been noted to occur during sleep in the able-bodied [31, 81-82], hence consciousness is not an absolute requirement. Rare cases of so-called true "spontaneous orgasm" have been described in the psychiatric literature where no obvious sexual stimulus can be ascertained [83] and which are different from the not uncommon "hyperesthesia sexualis" (orgasm following an extremely variable group of tactile, visual, auditory stimuli).

Exactly what initiates the orgasm has been a topic of discourse and speculation for many years. Four pseudo-neurophysiological models have been proposed. Sherfey's [84] model was based on the firing of stretch receptors in the pelvic striated muscles activated by the pelvic engorgement which initiated a spinal reflex. In Kaplan's model [85] the clitoris was the source for the sensory impulses activating the reflex. Mould's contribution [86-87] was to combine the Sherfey and Kaplan models and incorporate the gamma biasing of the muscle spindle of the striated musculature, which would then allow the generation of their clonic reflex contractions. Mould's hypothesized trigger was the dynamic stretch of the intrafusal fibers of the pelvic striated muscles via the alpha-fusimotor systems.

Davidson's model based on male ejaculation/orgasm [52] was grandly called the "Bipolar hypothesis" but it was in fact a "dual bipolar hypothesis". In brief, he proposed that when sexual arousal reached a critical level, a hypothetical central "orgasm centre" with upward links to the cortex and downward links to activate the smooth and the striated genito-pelvic musculature was triggered. The neural elements involved in seminal emission and in female uterine contractions fired to contract the smooth muscles and to inhibit arousal, while those involved in the contractions of the striated muscles caused the sensation of orgasm and its altered state of consciousness.

A more detailed exposition of the Davidson male model was attempted by Tuckwell [88] who explained the well-known refractory period for men after ejaculation/orgasm by a central build-up of neuronal extracellular K+. Women do not experience this inhibited arousability after orgasm presumably because they do not have the male emission/ejaculation mechanisms. Alternatively, the strong contractions of the uterus at orgasm are thought to be the comparable terminal event in females [52].

A more recent explanation is that the "switch off" is due to prolactin release. None of the models of orgasm initiation appear satisfactory [89]. While no new definitive mechanism(s) with laboratory-backed data has yet emerged, comparing brain imaging during female sexual arousal without orgasm [90-91] to brain imaging at orgasm offers at least the possibility of seeing whether there are any areas of the brain specifically involved in generating the orgasm.

C. PHYSIOLOGICAL ASPECTS OF WOMEN'S ORGASM

I. CENTRAL NERVOUS SYSTEM CONTROL OF WOMEN'S ORGASM

1. Overview

Studies of animals have provided some insights into the CNS control of sexual climax and are often the only avenue of information about the neural functions that coordinate the complex events leading up to and following orgasm. This section will first review the animal models of sexual climax and the brain areas that control genital reflexes. Because there has been very little research on genital function in female animals, studies in males are briefly described and comparisons with females are made. Then, studies of brain imaging during sexual arousal and orgasm are described and comparisons with the animal literature drawn. Next, information about brain disorders in humans is reviewed to provide insight into the function of relevant brain areas. Finally, brain areas that may mediate the inhibitory effects of antidepressant and antipsychotic medication are discussed, as well as the beneficial effects of drug and hormonal treatments.

2. Animal models of orgasm

There are a number of physiological markers that suggest the presence of orgasm in female animals, including increases in blood pressure and heart rate and uterine contractions during copulation in female rats, rabbits, cattle, and monkeys [92]. Much of the information about the physiological control of orgasm has come from studies of the urethrogenital (UG) reflex in male and female rats. This reflex, elicited by mechanical stimulation of the urethra or by electrical stimulation of certain brain areas, is characterized by a series of muscle contractions similar to those of orgasm in humans [93-94]. These contractions result from the coordinated firing of the pelvic, hypogastric, and pudendal motor nerves [93, 95]. In female rats this reflex includes rhythmic contractions of vaginal and uterine musculature as well as anal sphincter contractions [95].

Both the UG reflex in rats and orgasm in humans are thought to be controlled in part by a spinal pattern generator [92, 96-98]. Input to the pattern generator comes primarily from the sensory branch of the pudendal nerve; output is sent via pudendal motor neurons to the ischiocavernosus and bulbocavernosus muscles, the urethral and anal sphinctors, and striated muscles of the pelvis [99-101] and via hypogastric and pelvic nerves to sympathetic and parasympathetic preganglionic neurons.

3. Brain areas controlling orgasm

a) Tonic inhibition of the UG reflex

Animal studies have shown that genital reflexes are under tonic inhibitory control by the nucleus paragigantocellularis (nPGi) in the ventrolateral medulla. A majority (78%) of nPGi axons that project to the lumbosacral spinal cord contain serotonin (5-HT) [102], suggesting that the lumbosacral cord may be one site at which antidepressants of the selective serotonin reuptake inhibitor (SSRI) class act to inhibit orgasm in humans.

b) Excitatory influences on the UG reflex

The medial preoptic area (MPOA) is a major integrative site for the control of both male and female sexual behavior (reviewed in [103]). Electrical or chemical stimulation of the MPOA of anesthetized male or female rats elicited the UG reflex in the absence of genital stimulation and without spinal transection or lesions of the nPGi [92, 104]. Electrical stimulation also significantly decreased vaginal vascular resistance (increased engorgement) in anesthetized female rats and increased their blood pressure [105]. Thus, both sympathetic and parasympathetic influences were produced by MPOA stimulation, in accord with the integrated nature of the arousal and orgasmic response. In addition, one group of MPOA neurons fired during proceptive behavior of female rats and a different subset was active during lordosis [106]. Therefore, different neurons within the preoptic area appear to promote the female's sexual motivation and her receptive posture.

The MPOA does not send axons directly to the spinal cord but connects with the nPGi [107], which it presumably inhibits. In addition, the MPOA has reciprocal connections with the paraventricular nucleus (PVN) of the hypothalamus and the periaqueductal gray (PAG) of the midbrain [107-108]. The PVN is an integrative site for the sympathetic nervous system [109]. It also contains neurons that release oxytocin into the general circulation via the posterior pituitary and that project to the lumbosacral spinal

cord of male and female rats [110] and to the hippocampus [111]. Systemic oxytocin stimulates smooth muscle contractions, including those of orgasm.

The PAG consists of columns that subserve autonomic functions, pain perception, and female rat sexual behavior [112]. It receives input from the MPOA and from the area of the spinal cord in which the pudendal and pelvic nerves terminate [113], and it sends output that ultimately reaches the clitoris and penis [114-115]. The PAG also sends presumably inhibitory input to the nPGi [116]. Thus, the MPOA can inhibit the nPGi both directly and via its outputs to the PAG.

4. An orgasm-related circuit

The first study of brain activation in women during sexual arousal used blood-level-dependent functional magnetic resonance imaging (BOLD fMRI) during erotic or neutral visual stimuli [91]. All six women reported moderate sexual arousal in response to the erotic film, but not to the neutral film. Areas of greatest activation included the inferior temporal lobe, anterior cingulate gyrus, insular cortex, corpus callosum, thalamus, caudate nucleus, globus pallidus, and inferior frontal lobe. These areas are similar to those previously reported to be activated in men, although the men showed primarily unilateral activation [117].

A second study using BOLD fMRI compared activation in 20 female and 20 male undergraduates who were presented visual erotic or neutral stimuli [91]. Male students reported greater sexual arousal in response to the erotic film than did female students. Both male and female subjects showed increased bilateral activation in five cortical areas: the medial prefrontal cortex, the orbitofrontal cortex, the anterior cingulate cortex, the insular cortex, and the occipitotemporal cortex. In addition, both sexes showed bilateral activation of the amygdala and the ventral striatum. However, only males showed significant activation of the hypothalamus and the thalamus, although there was a nonsignificant trend toward activation of the hypothalamus in women. The only significant sex difference in activation was in the hypothalamus. However, when perceived sexual arousal was used as a covariate, the sex difference in hypothalamic activation was not significant. Thus, the lower level of perceived arousal in women was associated with lower hypothalamic activity. Several of the cortical and subcortical areas that were activated during sexual arousal have been associated with perception of emotional stimuli. These include the occipitotemporal (or inferior temporal) area, medial prefrontal cortex, and amygdala [118-120]. Activity in both the orbitofrontal cortex and ventral striatum have been associated with the presentation of rewards [121]. The anterior cingulate gyrus has been associated with autonomic and emotional processing [117] and goal-directed behavior [122].

The first studies of brain imaging (positron emission tomography, PET, coupled with MRI) during orgasm in women have recently been reported [123-124]. Two women with spinal cord injury (SCI) above the 10th thoracic segment (T10; i.e., at or above the level at which the hypogastric, pelvic, and pudendal nerves enter the spinal cord), as well as one non-injured woman, showed increased activation at orgasm, compared to pre-orgasm, of the paraventricular nucleus (PVN) of the hypothalamus, the central (or periaqueductal, PAG) gray of the midbrain, the amygdala, the hippocampus, anterior basal ganglia (striatum), cerebellum, and several regions of cortex, including the anterior cingulate, frontal, parietal, temporal, and insular cortices [124]. During self stimulation preceding orgasm, there was significant activation of the nucleus of the solitary tract (NTS, in the medulla), which receives sensory input from the vagus nerve, as well as of somatosensory and motor cortices, thalamus and sensory areas of the spinal cord and medulla. Several of the areas activated in women with SCI have previously been associated with orgasm, epileptogenic orgasmic auras, or sexual arousal in humans, including the prefrontal cortex (especially the right side: see [125]), anterior cingulate cortex [126], amygdala [126-129], and temporal and insular cortex [129].

A comparison of the areas activated by orgasm [124] with those activated during sexual arousal without orgasm [90-91, 124] reveals several differences. The most important appear to be the activation of the PVN, the PAG, the hippocampus, and the cerebellum with orgasm, but not with visual erotic stimuli. The PVN, as noted above, is an important integrative site for the sympathetic nervous system and supplies oxytocin to the peripheral circulation, via the posterior pituitary, and to the lumbosacral spinal cord. The central gray (PAG) receives and integrates autonomic input from the MPOA and PVN and appears to inhibit the nPGi in rats, thereby disinhibiting sexual reflexes. The roles of the hippocampus and cerebellum in elicitation of orgasm are unknown ***(Figure 3)***.

5. Central effects of drugs on women's orgasm

a) Serotonin

Selective Serotonin Reuptake Inhibitors (SSRIs) are

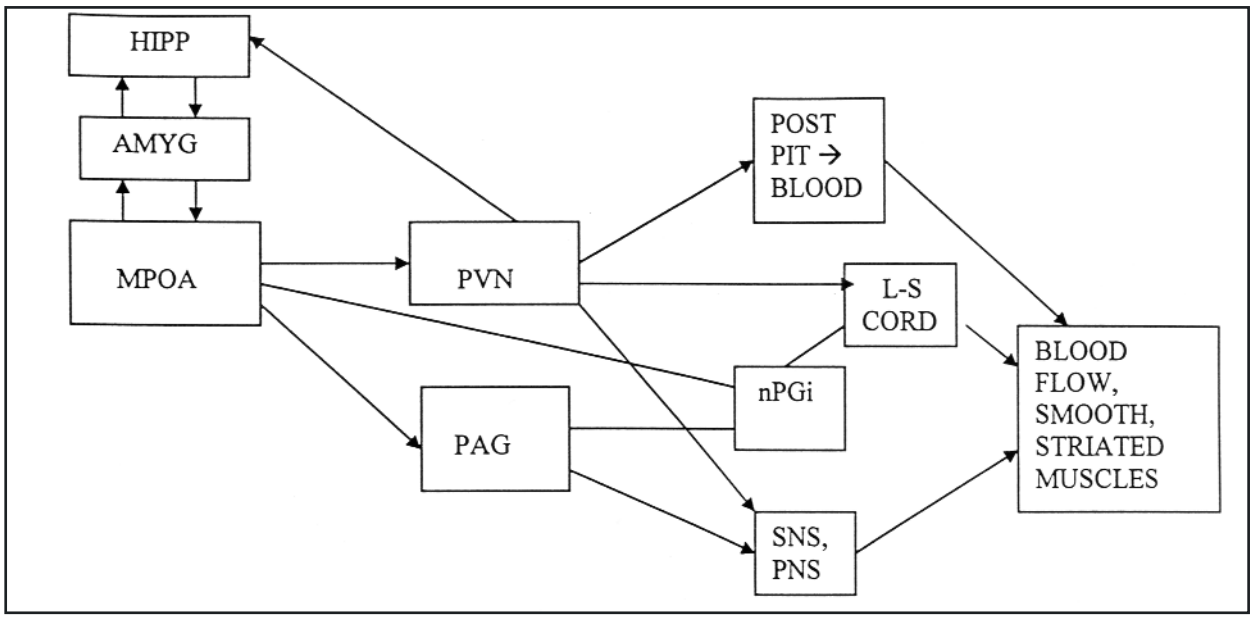

Figure 3 : Brain areas that affect orgasm. The medial preoptic area (MPOA) has reciprocal excitatory connections with the amygdala (AMYG), which in turn has similar connections with the hippocampus (HIPP). The MPOA also activates the paraventricular nucleus of the hypothalamus (PVN) and the periaqueductal gray (PAG, or central gray), but inhibits the nucleus paragigantocellaris (nPGi) of the medulla, which, in turn, inhibits the orgasm control area in the lumbosacral spinal cord (L-S CORD). The PVN sends excitatory oxytocinergic axons to the hippocampus (HIPP), the L-S CORD, and the posterior pituitary (POST PIT); the oxytocin is released from the POST PIT into the general circulation and stimulates smooth muscle. The PAG activates components of the sympathetic and parasympathetic nervous systems (SNS, PNS) and also inhibits the nPGi. Thus, the MPOA, both directly and via the PAG, can inhibit the nPGi, and thereby disinhibit the L-S CORD. The L-S CORD, the SNS, and oxytocin from the POST PIT trigger striated and smooth muscle contractions and alter patterns of blood flow to the genitals. In addition, the anterior basal ganglia, cerebellum, and several regions of cerebral cortex (anterior cingulate, frontal, parietal, temporal, and insular cortices) are activated by orgasm, although the anatomical pathways interconnecting those areas are not well understood.

noted for their inhibitory effects on orgasm/ejaculation and libido (reviewed in [130]). However, fewer inhibitory side effects have been reported with some antidepressants than with others. For example, bupropion produced fewer adverse sexual effects in both men and women than did SSRIs [131-134]. (See **Table 4** for a summary of effects on orgasm of centrally acting drugs.) Bupropion is a weak inhibitor of serotonin and norepinephrine transport and a more potent inhibitor of dopamine transport, as well as an agonist at 5-HT1A receptors; both the increase in extracellular dopamine and the stimulation of 5-HT1A receptors may explain its lower incidence of sexual side effects [135]. Furthermore, this profile of effects suggests a similarity with the facilitation of ejaculation in male rats and monkeys by 5-HT1A receptors, rather than the inhibition of the lordosis posture in female rats by that receptor subtype. Nefazodone also has a lower incidence of sexual side effects, perhaps because it is a 5-HT2 antagonist, as well as an SSRI [136-138]. Stimulation of 5-HT2 receptors has been reported to inhibit the release of both norepinephrine and dopamine from several brain areas (reviewed in [139]). Because dopamine and norepinephrine facilitate sexual behavior (see below), the increase in serotonergic activity at 5-HT2 receptors could explain some of the inhibitory effects of SSRIs on orgasm [139-140]. Moclobemide (a monoamine oxidase A inhibitor, which would increase levels of both 5-HT and norepinephrine) and amineptine (a dopamine transport inhibitor) are also antidepressants with a lower incidence of anorgasmia than the SSRIs [137, 141-142]. Indeed, there is a single case report of hyperorgasmia in a woman taking moclobemide [143]. Therefore, the coordinated increases in either norepinephrine or dopamine appear to offset the inhibitory effects of serotonin on orgasmic ability.

Noradrenergic activity may also improve the profile of effects of the antidepressant mirtazapine. It inhibits a2 autoreceptors on norepinephrine terminals and also a2 heteroceptors on 5-HT terminals [144]. As a result both norepinephrine and 5-HT levels are increased. It is also an antagonist at postsynaptic 5-HT2 and 5-HT3 receptors [144]. A prospective, 12-week, open-label trial with 18 women reported a 48% improvement of ease and satisfaction with

orgasm [145]. However, 50% of the participants dropped out of the study, suggesting that other side effects outweighed the improved orgasmic ability in some women.

Among the typical SSRIs there may also be differences in inhibition of orgasm. Paroxetine delayed orgasm more than fluvoxetine, fluoxetine, and sertraline [141] and more than nefazodone, fluoxetine and venlafaxine [138]. One explanation for this greater impairment may be that paroxetine is a more potent inhibitor of the serotonin transporter than are fluoxetine and fluvoxetine, and does not inhibit the dopamine transporter, as does sertraline and, to a lesser degree, fluoxetine and fluvoxetine (reviewed in [130]). As noted below, dopamine antagonists impair several aspects of sexual function, whereas the dopamine precursor, L-Dopa, is facilitatative. Both women and men treated with fluoxetine, paroxetine, and sertraline for anxiety disorders reported delays in reaching orgasm and decreased quality of orgasm at one- and two-month follow-ups [146]. However the impairments in the fluoxetine group (but not the other two groups) had abated by the end of the third month. There have been some reports of little or no effect of SSRIs on orgasm. Indeed, one multi-center open-label study of fluoxetine, conducted by the manufacturer, reported an improvement in women's orgasmic ability, associated with improvement in depressive symptoms [147]. Similarly, a small pilot study found no effect of fluoxetine on orgasm [148], and two industry-sponsored trials found either no effects of fluoxetine or clomipramine on orgasm, or that there were too few complaints to analyze statistically (reviewed in [149]).

Several factors may account for the variability of response to a given drug. First, there are individual differences in the numbers and anatomical distributions of the relevant receptor subtypes. Thus, increases in extracellular serotonin, resulting from inhibition of uptake, may activate different ratios of 5-HT2 and 5-HT1A receptors in different people. There may also be individual differences in the inhibition of dopamine and norepinephrine release by 5-HT2 receptors. In addition, improvements in general and interpersonal functioning may tend to offset perceptions of sexual impairment in some women. A major factor in assessing the effects of drugs on orgasm is the method of questioning the subjects. Retrospective reports are less reliable than are daily logs.

b) Dopamine

The effects of dopaminergic drugs in humans parallel those reported in animals [150-155]. Although most reports on the effects of antipsychotic drugs have studied male patients, women patients also reported inhibited orgasm from the antipsychotic drugs trifluoperazine, fluphenazine, and thioridazine [156 ; reviewed in 157]. Antipsychotic-induced sexual dysfunction may result directly from blockade of dopamine receptors in areas critical for sexual function, such as the MPOA and PVN, or indirectly from increased prolactin levels, extrapyramidal side effects, or sedation. One open-label ongoing-treatment study found no effect of either haloperidol or clozapine on orgasmic ability ; however, the authors noted that the women may have under-reported sexual side effects [158].

c) GABA

A retrospective clinical study of women taking antiepilepsy drugs (primarily benzodiazepines) reported that women with epilepsy who were taking antiepileptic drugs found orgasm less satisfying than did the healthy, unmedicated controls [159]. Untreated women with epilepsy were not significantly different from either of the other two groups. In the treated women antiepileptic drugs increased both total testosterone and steroid hormone binding globulin, but did not affect levels of free testosterone, compared to the other two groups. Thus, alterations in free testosterone cannot explain the impairment in orgasmic enjoyment.

d) Nitric oxide

Sildenafil (Viagra) selectively inhibits phosphodiesterase V, and thereby prolongs the vasodilatory effect of nitric oxide. Although sildenafil has been used successfully for treatment of erectile dysfunction in men, there have been mixed reports of the effects of sildenafil on women's orgasmic function. Caruso et al. [160] found improved sexual arousal and orgasm with sildenafil. However, a minority of women responded positively in three other studies [161-163].

e) Norepinephrine/epinephrine

A retrospective study of 1080 women, who responded via a self-administered questionnaire, reported no significant increase in difficulty achieving orgasm while taking hydralazine, beta-adrenergic antagonists or methyldopa [164]. Similar results were obtained in a prospective randomized double-blind study of 345 women over a period of 24 months [165] and in unmedicated healthy controls [166]. Therefore, drugs that inhibit beta-adrenergic receptors appear not to affect women's orgasmic ability.

However, SSRI-induced inhibition of norepinephrine release onto a1 receptors could explain some of the impairment of orgasm, as noted above.

f) Acetycholine

As in female rats [167], atropine failed to affect blood flow in women during sexual arousal and also did not affect subjective sexual arousal or orgasm [168]. Therefore, acetylcholine appears to play a minor role in the control of vaginal blood flow, sexual arousal, and orgasm.

g) Estrogens

There is little evidence of estrogenic facilitation of orgasmic function in women. In an uncontrolled, open-label trial, 25% of 188 premenopausal women reported an improvement in orgasmic ability [169]. However, a retrospective study of 66 women who were oophorectomized and hysterectomized found no difference in orgasmic ability between the 33 who received conjugated estrogens and the 33 who did not [170]. Similarly, two prospective studies of postmenopausal women found little effect of estrogen plus progestin hormone replacement therapy (HRT) [171-172]. Each compared HRT to tibolone, which can be metabolized into estrogenic, androgenic, and progestogenic compounds. The former, single-blind study of 50 women found no effect of either treatment, based on a questionnaire at baseline and after one year. The latter, open-label study of 48 women found a significant improvement after three months of tibolone treatment, but not HRT.

h) Androgens

A prospective, three-month, open-label study of 44 oophorectomized and hysterectomized women found that a monthly injection of estrogen and testosterone (E+T) increased the rates of orgasm during the first three weeks after the injection [173], compared to these women's own baseline and compared to E-alone or no treatment. The E+T-treated women had been receiving monthly hormone injections for up to two years, but had not received an injection for 8 weeks prior to the baseline measure. At the time of the baseline interview and hormone sample these women reported relatively low rates of coitus and orgasm, although their T levels were at least four times the normal levels of gonadally intact premenopausal women (based on normal ranges from Endocrine Sciences reported in [174]). Therefore, the T from the previous injection 8 weeks before baseline measures had been metabolized rather slowly. These data suggest that, although extremely high levels of T can improve sexual interest and orgasmic ability, more moderate levels have little effect.

A more extensive and well-controlled study of 75 oophorectomized and hysterectomized women found similar results [174]. Conjugated estrogens were administered either alone or with 150 or 300 mg T per day in transdermal patches. The higher dose of T improved orgasm pleasure. However, again the effective T levels were two to six times the normal range in intact women. Finally, 113 women with low levels of both T and dehydroepiandrosterone (DHEA) and complaints of orgasmic difficulty were treated for at least three months with DHEA (50 mg/day orally) [175]. These women reported a greater frequency of orgasm after treatment than before. Their T and DHEA levels were in the upper half of the normal range at the end of the three-month treatment. Unfortunately, there was no control treatment, and the study was open-label. A possible mechanism or androgenic facilitation of sexual function is a decrease in sex hormone binding globulin, which would increase levels of free estradiol and testosterone **(Table 4)**

II. SPINAL CORD PATHWAYS INVOLVED IN WOMEN'S ORGASM

Our ability to assess the impact of spinal lesions on orgasm in humans is unique because spinal cord lesions are relatively easily located and described via detailed neurologic exam. Thus, the subject of female orgasm and the impact of spinal cord injuries (SCIs) or damage has received significant attention in recent years and the level and degree of evidence for this phenomenon has increased greatly. Once flippantly considered "phantom" [176], the orgasmic experiences of women with SCIs have recently been described through multiple controlled studies that have consistently documented the existence of orgasm in women with SCIs. Moreover, these studies have begun to describe the attributes of orgasms in women with SCIs. Table 5 describes the notable studies that have assessed the impact of orgasm in humans with SCIs. As the studies are numerous, only details regarding those reports that are most significant will be discussed **(Table 5)**.

In the early 90's interest in the impact of SCIs on female sexuality and sexual response reemerged after previous discussion of "phantom orgasms". During this time, a number of questionnaire studies

Table 4 . Effects of Centrally Active Drugs on Orgasm

Authors	Design	N	Drugs	Conclusion	Level of evidence
I. Antidepressants					
Shen & Hsu (1995) [131]	Retrospective reports	110 women	Fluoxetine, paroxetine, sertraline, bupropion	Few adverse effects with bupropion; similar inhibition of orgasm with the other 3	Clinic records
Lauerma (1995) [143]	Retrospective reports	1woman	Moclobemide	Hyperorgasmia with this MAO-A inhibitor (increased 5-HT and NE)	Single case, patient report
Feiger et al. (1996) [136]	Prospective, randomized; 6 wks	143 men & women	Sertraline, nefazodone	Sertraline impaired orgasm in both men & women equally; nefazodone had no negative effects	Sexual function questionnaire
Montejo-Gonzalez et al. (1997) [141]	Prospective, multicenter, open label; 17 mo	192 women, 152 men	Paroxetine, fluvoxetine, fluoxetine, sertraline, moclobemide, amineptine	Paroxetine delayed orgasm more than fluvoxetine, fluoxetine & sertraline; few adverse effects with moclobemide or amineptine; women more severely affected; men more frequently affected	Descriptive, physician questioning
Modell et al. (1997) [132]	Retrospective; open-label; patient population	57 women, 49 men	Bupropion, fluoxetine, paroxetine, sertraline	Greater duration and intensity of orgasm with bupropion; n.s. trend toward increased time to orgasm; fluoxetine and paroxetine decreased orgasm intensity, increased time to orgasm, and produced a n.s. trend to decreased duration of orgasm; sertraline decreased duration of orgasm, increased time to orgasm, and produced a trend to decreased intensity of orgasm	Descriptive; anonymous questionnaires mailed to patients in practices taking antidepressants; variable return rate

Table 4 . Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusion	Level of evidence
Piazza et al. (1997) [148]	Prospective; open-label; 6 wks; no placebo	14 women, 11 men	Sertraline, paroxetine	No change in orgasm in women; other aspects of sexual function improved; impaired orgasm in men	5-item self-report scale, administered before and after 6 wk tx
Kavoussi et al. (1997) [133]	Prospective randomized, double-blind, parallel group; 16 wks	119 women, 129 men	Bupropion, sertraline	Orgasm dysfunction more common with sertraline compared to bupropion	Investigator-conducted interview at each office visit
Labbate et al. (1998) [146]	Prospective; 3-mo	19 women, 12 men w/ anxiety disorder; 18 women, 12 men w/ de-pression	Fluoxetine, sertraline, paroxetine	Decreased quality and longer delay in orgasm compared to baseline; anorgasmia more common in women than in men; similar effects of all 3 drugs on both patient groups at all 3 times	Descriptive, monthly rating on visual analog scales
Boyarsky et al. (1999) [145]	Prospective, open-label, flexible dosing; 12 wks	18 women, 7 men	Mirtazepine	Ease/satisfaction with orgasm improved 48% with mirtazepine compared to baseline; 50% dropout rate	Questionnaire administered bimonthly
Kennedy et al. (2000) [142]	Prospective, 8 or 14 wks	65 women, 42 men	Paroxetine, sertraline, moclobemide, venlafaxine	No difference between women and men in orgasm impairment; paroxetine and sertraline produced more dysfunction than moclobemide and vinlafaxine in women	Sexual Functioning questionnaire before and after antidepressant
Coleman et al. (2001) [134]	Multicenter, randomized double-blind; 8 wks	288 women, 168 men	Bupropion SR, fluoxetine	~ 30% of fluoxetine-treated patients had orgasm dysfunction; bupropion SR- and placebo-treated patients had ~ 10%.	Weekly questioning by physician

Table 4. Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusion	Level of evidence
Montejo et al. (2001) [137]	Multicenter, prospective open-label; total period 5 yrs (variable time per subject, from <3 mo to >1 yr)	610 women, 412 men ; normal sexual function before tx	Fluoxetine, sertraline, fluvoxetine, paroxetine, citalopram, venlafaxine, mirtazapine, nefazodone, amineptine, moclobemide	All SSRIs and venlafaxine produced more delayed orgasm or anorgasmia than mirtazapine, nefazodone, amineptine, or moclobemide; women had fewer but more severe dysfunctions than men	Psychotropic-Related Sexual Questionnaire at each office visit before and during tx
Michelson et al. (2001) [147]	Multicenter, prospective; Acute phase: open-label 13 wks; Continuation phase: randomized, double -blind, 25 additional wks	342 women, 159 men	Fluoxetine	Acute phase: 44% of women reported improvement in orgasmic ability, 38% reported no change, 18% reported orgasmic impairment; both improvement and impairment in orgasmic ability associated with improvement and impairment, respectively, in depressive symptoms	Self-rated, 4-question before, at end of acute phase, and weekly in continuation phase; study conducted by Eli Lilly, manufacturer of fluoxetine
Bobes et al. (2002) [138]	Prospective open-label; 6 mo tx	58 women, 43 men	Nefazodone, fluoxetine, paroxetine, venlafaxine	Orgasm improvement with nefazodone and orgasm impairment with paroxetine in in women	Semistructured interview before and after treatment; funded by Bristol Myers Squibb, manufacturer of nefazodone
II. Antipsychotics					
Ghadirian et al. (1982) [156]	Retrospective open-label, ongoing tx	29 women, 26 men; random sample of outpatients	Fluphenazine	33% reported decreased quality of orgasm; 22% reported decreased ability to achieve orgasm; impairment in men, but not women, associated with elevated prolactin	Authors' questionnaire

Table 4 : Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusion	Level of evidence
Hummer et al. (1999) [158]	Prospective open-label, ongoing tx; assessed weekly during 1st 6 wks & monthly thereafter	37 women, 116 men	Haloperidol, clozapine	0/12 women taking haloperidol reported orgasmic dysfunction, compared to 8 /41 men (19.5%); 1/25 women (4%) taking clozapine reported orgasmic dysfunction, compared to 17/25 men (22.7%); fewer women reported dysfunction; no difference b/w drugs; authors note women may have under-reported effects	Observer-rated side effect rating scale
III. Phosphodiesterase inhibitors					
Kaplan et al. (1999) [161]	Open-label nonrandomized; 12-wk	30 postmeno-pausal women	Sildenafil	Orgasm satisfaction improved 7.4%	Self-administered questionnaire
Caruso et al. (2001) [160]	Prospective souble-blind, crossover; 12 wks	51 premeno-pausal women; arousal disorder	Sildenafil	25 and 50 mg sildenafil increased orgasm frequency, compared to placebo and baseline; placebo increased orgasm relative to baseline	Self-administered questionnaire, once/wk
Berman et al. (2001) [163]	Prospective open-label; 6 wks	7 women w/ history of sexual abuse; 24 w/o	Sildenafil	2/7 w/ history of childhood sexual abuse (CSA) reported improved orgasmic ability; 19/24 w/o CSA improved	5-item questionnaire at end
Berman et al. (2001) [162]	Prospective open-label; 6 wks	48 women; Sexual Arousal Disorder (SAD)	Sildenafil	67 % improved orgasmic ability	Questionnaire at baseline & end of study; psychological measures

Table 4. Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusion	Level of evidence
IV. Antihypersensives					
Bulpitt et al. (1989) [164]	Retrospective questionnaire	1080 women, 1285 men	Hydralazine, beta-adrenergic antagonists, methyldopa	Women showed no increased difficulty achieving orgasm with any of the drugs	Self-administered questionnaire
Grimm et al. (1997) [165]	Prospective randomized, controlled, double-blind; 48 mo	345 women, 557 men	Acebutolol, amlodipine maleate, chlorthalidone, doxazosin maleate, enalapril maleate	Women showed no increased difficulty difficulty achieving orgasm with any of the drugs	Questioning by physician at baseline and annually during tx
Duncan et al. (2000) [166]	Ambulatory medical record-based choice of subjects; case-control	104 mildly hypertensive women, 107 unmedicated healthy controls	ACE inhibitors, _ adrenergic blockers, Ca^{2+} channel blockers, diuretics, combination drugs	No difference b/w medicated and unmedicated hypertensives; impaired orgasm in hypertensives compared to healthy women; less orgasm frequency in smokers compared to nonsmokers (not associated with age or hypertension)	Self-administered questionnaire and phone interview
V. Anticonvulsants					
Duncan et al. (1997) [159]	Retrospective	243 women	Anti-epilepsy Drugs (AED)	159 epileptic women taking AEDs: less orgasm satisfaction compared to 48 healthy controls; no difference in no tx epileptic women	Validated questionnaire & testosterone assay
VI. Anticholinergics					
Wagner & Levin (1980) [168]	Controlled laboratory study	11women	Atropine, Methylatropine	Neither tx affected orgasm or vaginal blood flow; muscarinic cholinergic receptors do not appear important for orgasm or blood flow	Controlled laboratory study

Table 4 . Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusions	Level of evidence
VII. Estrogens					
Nathorst-Boos et al. (1993) [170]	Retrospective clinical study	66 oophorectomized, 35 hysterectomized w/ ovaries	Conjugated estrogens (ERT)	No improvement in orgasmic ability in 33 oophorectomized women, compared to 33 oophorectomized no-ERT women	Questionnaire and structured interview
Eicher & Muck (1996) [169]	Prospective open-label; 4 mo	188 women with sexual dysfunction	Estradiol transdermal patch	25% improved orgasmic ability	Uncontrolled clinical study
Kokcu et al. (2000) [171]	Prospective single-blind; 1 yr	50 postmenopausal women	Conjugated estrogens + medroxyprogesterone acetate (HRT), tibolone	No improvement in orgasm frequency (tibolone has estrogenic, androgenic & progestogenic metabolites)	Questionnaire at baseline and after 1 yr
Wu et al. (2001) [172]	Prospective open-label; 3 mo	48 postmenopausal women	HRT, tibolone	Tibolone improved orgasmic ability compared to HRT	Questionnaire at end of 3 mo
VIII. Androgens					
Sherwin & Gelfand (1987) [173]	Prospective open-label; 1 mo (no hormone injection 8 wks pre-baseline)	44 oophorectomized & hysterectomized women	E (8.5mg) + T (150mg), E alone (10 mg), no tx	Orgasm and coitus rates higher in E+T group during 1st 3 wks after monthly injection, compared to E or no tx controls and compared to baseline	Daily recording & hormone assays at , baseline and days 2, 4 8, 15, 21, & 28 of tx
Shifren et al. (2000) [174]	Prospective double-blind, counterbalanced; 9 mo	75 oophorectomized + hysterectomized women	Conjugated E plus either T (150 or 300_g/d transdermal) or placebo	300_g/d of T improved orgasm pleasure and frequency of sexual activity	Questionnaire & sexual function and diary completed by phone

Table 4. Effects of Centrally Active Drugs on Orgasm (Ctd)

Authors	Design	N	Drugs	Conclusion	Level of evidence
Munarriz et al. (2002) [175]	Retrospective open-label, clinical tx; min 3 mo	113 women w/ low T & dehydroepi-androsterone (DHEA)	DHEA (50mg/d)	Greater orgasm frequency after DHEA tx; both DHEA and T increased to upper range of normal female levels	Questionnaires & blood samples for hormone levels

Table 5 . Studies of Orgasm in Women with Spinal Cord Injuries

Author	Design	N	Conclusion	Level of Evidence
Money (1960) [176]	Interview of hospitalized patients, no neurologic data	7 women with SCI, no controls	Coined the term "phantom orgasm" because patients reported orgasms in their sleep	Case Report
Siosteen (1990) [177]	Structured questionnaire administered via interview, neurologic examination	13 women with SCI, no controls	33% unaltered orgasm, 25% orgasm decreased, 42% absent, 50% reported interest was unchanged, 33% interest decreased and 17% reported no interest	Uncontrolled questionnaire
Kettl (1991) [178]	Mailed questionnaire, subjects reported neurologic data	74 mailings to women with SCI, 27 responses	52% orgasmic, 7 reported different orgasm, 3 exactly the same, 2 similar; 25% complete paraplegics and 25% complete quadriplegics orgasmic; women with complete quadriplegia related frequently of sexual activity as much less than others	Pre/post questionnaire
Charlifue et al. (1992) [179]	Telephone survey with neurologic data	293 subjects identified, 231 SCI women, No controls	"Approximately half of the women reported they had experienced orgasm since their injuries; stimulus generally genital or genital combined with breast"	Uncontrolled questionnaire
Sipski & Alexander (1993) [180]	In person 80 item questionnaire, neurologic data provided	25 women with pre/post results; pre/post controls	44% reported ability to have orgasm; ability was not related to the degree of injury; frequency and satisfaction with sexual activity significantly decreased	Pre/post questionnaire

Table 5. Studies of Orgasm in Women with Spinal Cord Injuries (Ctd)

Author	Design	N	Conclusion	Level of Evidence
Harrison (1995) [181]	Mailed questionnaire, no neurological data	Sent to 226 women, 85 answered	Pre injury 46% were orgasmic, post injury 36% were orgasmic, 7% were not orgasmic pre injury compared to 23% post, 33% reported this question was NA pre injury and 12% post, 14% did not pre injury and 28% did not post, frequency 55.6% report greater than once per week pre injury compared to 38% post	Pre/post questionnaire
Sipski et al. (1995) [184]	Laboratory based analysis along with detailed American Spinal Injury Association (ASIA) exam, EMG, and SSEP, SCI questionnaire and DSFI; subjects self-stimulated (max 75 min) in any way they desired to orgasm; HR, BP, RR, rectal contractions, quality of and time to orgasm monitored	25 SCI subjects at T6 and above, 10 age-matched able-bodied controls	52% of SCI subjects achieved orgasm; degree and type of SCI did not affect ability to achieve orgasm; orgasmic subjects higher on the sexual information and sex drive subset of the DSFI	Controlled, laboratory study with physiologic testing
Kreuter (1996) [182]	80-item questionnaire; subjects contacted by phone then completed a mail in questionnaire, neurologic data was included	264 subjects, 167 SCI and 97 age- and sex-matched randomly selected controls; 59% had partner	67% of SCI seldom or never experienced orgasm compared to 6% of controls	Controlled questionnaire study

Table 5 . Studies of Orgasm in Women with Spinal Cord Injuries (Ctd)

Author	Design	N	Conclusion	Level of Evidence
Whipple et al. (1996) [186]	Laboratory based study along with American Spinal Injury Association (ASIA) exam; subjects performed vaginal, cervical and hypersensitive area stimulation using fixed methodology; BP, HR, anxiety, sexual arousal, endogenous pain, and spasticity recorded	16 complete SCI at T6 and below, 5 able-bodied controls	Three SCI subjects and 1 able-bodied subject reported orgasms during the study	Controlled, laboratory-based study
Jackson (1999) [183]	Pre-post injury questionnaire with neurologic data	478 subjects; multicenter study; 315 sexually active	54% reported orgasms, of those who were orgasmic 32% of injuries were cervical, 41% thoracic and 52% lumbosacral; 32% had complete injuries and 41% had incomplete injuries	Pre-post questionnaire, multi-center study
Sipski et al. (2001) [189]	Laboratory-based study coupled with detailed neurologic assessment, SCI questionnaire, DSFI; HR, BP and RR monitored	69 SCI, 21 able-bodied controls; all levels of SCI represented	44% of SCI subjects were orgasmic in the laboratory vs 100% of controls; subjects with complete lower motor neuron injuries affecting the sacral spinal segments were significantly less likely to achieve orgasm than all other SCI subjects	Controlled, laboratory-based study with physiologic testing

were published with larger sample sizes [177-182] and accompanying neurologic data was often included [177-180, 182]. Although some studies still suffered from a lack of controls [177, 179], others included pre-post injury data as a form of control [178, 180-181]. These studies documented that women with SCIs did experience orgasms and that, in general, approximately 50% of the women noted the ability to attain orgasm was present post-injury. Similar findings were also noted in the largest study of females with SCI to date. In a multicenter study, Jackson [183] reported on 478 subjects. Of these subjects, 315 were sexually active since their injuries and of this subgroup 54% reported achieving orgasm post injury.

The next advance in the study of orgasm in women with SCIs has been the introduction of the laboratory-based assessment of women's orgasmic capacities. Sipski et al. [184] studied 25 women with SCIs at and above the level of T6 using standardized criteria [185] and compared them with 10 age-matched, able-bodied control subjects. Subjects were given 75 minutes to perform self-stimulation to orgasm in any way they chose. All able-bodied subjects achieved orgasm as compared to only 52% of SCI subjects. Degree and type of SCI were not found related to the SCI subject's ability to achieve orgasm. Orgasmic SCI subjects scored higher on the sexual information and sexual drive components of the Derogatis Sexual Functioning Inventory (DSFI).

Sixteen women with SCIs at and below the level of T6 were studied along with 5 able-bodied control subjects [186]. Subjects used a modified tampon to stimulate their cervix and vagina at monitored and specified levels of pressure. Three subjects with complete SCIs and one able-bodied subject had orgasms under these conditions ; one for the first time. Based upon these data and other animal reports [187-188] the authors hypothesized that the vagus nerve conveys a sensory pathway from the cervix to the brain that is responsible for the preservation of the ability to achieve orgasm in women with SCIs.

More recently, Sipski et al. [189] expanded their study of orgasm to include women with all levels of injuries. Identical methodology was used to a previous study [185], thus data were combined. A total of 66 women with SCIs and 21 able-bodied controls were examined. Women with complete lower motor neuron injuries affecting their S2-S5 reflex arc were significantly less likely than other subjects to achieve orgasm. Overall, 55% of all SCI subjects reported orgasmic ability post-SCI whereas 44% were orgasmic in the laboratory. Subjects with SCIs took significantly longer (26.37 minutes) than able-bodied subjects (16.33 minutes) to achieve orgasm. Blood pressure, heart rate, and respiratory rate responses were similar between able-bodied and SCI subjects throughout the study. Moreover, subjective descriptions of sensations during orgasm were indistinguishable between able-bodied and SCI subjects. These authors reported the importance of an intact sacral reflex arc in the ability to achieve orgasm. Moreover, the presence of the urethrogenital reflex in spinalized rats [95, 104] that mimics orgasm in humans provides an animal model that is consistent with this hypothesis.

Overall, there is a strong level of evidence for the occurrence of orgasm in women with SCIs. There is also substantial evidence of the impact of specific injuries on orgasmic potential. Future human and animal studies are warranted to confirm the specific effects of spinal lesions on the ability to achieve orgasm. Women with spinal disorders other than SCIs would also be appropriate to study.

III. PERIPHERAL NERVOUS SYSTEM INVOLVEMENT IN WOMEN'S ORGASM

In brief, the nervous supply of the genitalia is by the sympathetic and parasympathetic branches of the autonomic nervous system pelvic nerves, hypogastric nerve, paravertebral sympathetic chains and by somatic nerves (pudendal nerve from the pelvic splanchnic branches and sacral plexus). The nerves are either efferents that convey nervous impulses from the brain and spinal cord to control motor, secretory and vascular functions, or afferents that mediate sensation, usually by specialized nerve endings. The autonomic nerves regulate blood flow and the involuntary smooth muscle while the somatic nerves control the voluntary or striated muscles. Sensory nervous traffic can be mediated by both the somatic and autonomic systems. The nerves release a number of neurotransmitters, classically nor-adrenaline at the sympathetic nerve endings and acetylcholine at the parasympathetic and somatic. However, the former two systems become mixed in the pelvic plexus and with the recognition of NANC (non-adrenergic, non-cholinergic) nerves, many different transmitters and neuropeptides exist and are co-localized. Much of this knowledge comes from animal studies, usually rodent (rabbit /rat) studies; genital

data from women is sparse. While the studies using immunohistochemical techniques to identify and localize the various neurotransmitters give some insight as to what structures are innervated and by what chemicals, they unfortunately do not give any information of the exact functions of the nerves/neurotransmitters and therefore some degree of informed speculation has to be applied. Their proposed actions, together with those of secreted hormones, bring about the peripheral changes observed in sexual arousal and at orgasm.

1. Vagina

The anterior wall, the area with the highest erotic sensitivity, has a denser innervation than the posterior wall and the distal area has more nerve fibers than the proximal [190]. According to Krantz [34] aggregated ganglion cells and nerve fibers were present in the adventitia surrounding the vagina. The fibers, filiform in shape, penetrated and supplied the muscularis and larger blood vessels. Hoyle, Stones, Robson, Whitley & Burnstock [191] showed that nerves were also closely applied to the papillary capillaries and were possibly "sensorimotor" nerves (sensory nerves stimulated by antidromic impulses giving them an efferent function).

The changes induced by sexual arousal for the vagina are summarized diagrammatically in Figure 3. Briefly, the unaroused human vagina has a low (acid) surface pH, minimal surface fluid, low blood flow and low surface p02. A good sympathetic tone is probably maintaining this low flow by its constricting effect on the blood supply. Oral administration of the alpha-adrenoceptor blocker phentolamine to premenopausal women increased their vaginal blood flow indicative of a basal constrictive adrenergic tone, but whether the drug acted centrally or peripherally or both is unclear [192].

Effective sexual arousal causes a rapid increase in the blood flow activated by the release of VIP (Vasoactive Intestinal Peptide) from NANC-nerve endings, while NPY (Neuropeptide Y) probably constricts the venous drainage creating engorgement. The increased hydrostatic pressure in the capillaries forces a protein poor, plasma-like fluid into the tissues spaces (= tissue fluid) which then percolates through the Na+-absorbing epithelium onto the surface of the vagina as the increased surface fluid lubrication [35]. This neurogenic transudate (pH~7.4) can partially neutralize the acidity of the basal surface vaginal fluid (ph~4-6) and thus raise the vaginal pH [193]. The enhanced blood flow increases the vaginal surface p02 facilitating the use of aerobic rather than anaerobic mechanisms to generate energy by any sperm when ejaculated into the vagina (see [1, 35, 194] for references). Laan et al. [195] provided evidence for a facilitatory effect of sildenafil (an inhibitor of phosphodiesterase 5, PDE5 influences male genital blood flow by controlled the level of cyclic GMP and nitric oxide [NO]) on vaginal photoplethysmograph measures of sexual arousal. Meston & Worcel [196] demonstrated a beneficial influence of the nitric oxide precursor L-arginine in combination with the alpha2 blocker yohimbine on genital engorgement in postmenopausal women. These findings lend support for a role of NO in the enhancement of vaginal blood flow, although very little NOS has been found in the vagina using immunohistochemistry [191].

Meston and colleagues provided evidence for a facilitatory role of peripheral adrenergic activation on sexual arousal in women. Ephedrine (50mg), an alpha and beta adrenergic agonist, facilitated vaginal photoplethysmograph measures of sexual arousal [197], and clonidine, an alpha2 adrenergic agonist which blocks peripheral sympathetic outflow, decreased these responses [198]. Increased sympathetic nervous system activity, induced via intense acute exercise, enhanced vaginal engorgement in women [199-201].

The pattern and density of innervation of the vaginal vasculature and microvasculature was described by Hoyle et al [191] using immunohistochemistry in surgical specimens taken from five pre- and five postmenopausal women. They identified a number of neuropeptides in the papillae, subepithelial plexus, propria arteries and veins and the deep arteries and veins. These included Neuropeptide Y (NPY), Vasocactive Intestinal Peptide (VIP), CGRP, Substance P (SP) and the enzyme NOS. The vasomotor properties of VIP (vasodilatation), NO (from NOS production- vasodilatation) and NPY (vasoconstriction) are well-known but the neuropeptides CGRP, NPY and SP (and also NO and VIP) are known to be involved in sensory nerve function and can influence the permeability of the capillaries. Orgasm is presumed to cause the decreased release of all of these and to enhance the release of the adrenergic system transmitters, thus effectively decreasing the blood flow and the production of vaginal lubrication. At present however, it should be said that our knowledge of the exact functions of these active agents lags far behind our knowledge of their locations.

Less attention has been paid to the longitudinal and circular smooth muscle coats of the vagina. They can contract spontaneously even in the non-pregnant woman and especially around menstruation, although the contractions are not perceived in consciousness [1, 202-203]. The muscles possess both alpha and beta adrenoreceptors: blockade of the alpha system inhibits spontaneous contractions of vaginal muscle strips while beta blockade induces greater adrenergic-mediated contractions [204]. The vipergic innervation (neurotransmitter = VIP) decreases both tone and induces relaxation. During arousal, the vipergic innervation is likely to be dominant by facilitating smooth muscle relaxation of the vaginal wall and thus not reducing the caliber of its blood vessels, thereby allowing the VIP-activated lubrication mechanism to operate. Contraction of the vaginal smooth muscle if it happens does not occur until the late excitation state just before orgasm ***(Figure 4).***

2. Labia minora

Although the neural mechanisms and the neurotransmitters creating the congestion and increased blood flow responses of the labia minora to sexual arousal have not been characterized, they are probably mediated through mechanisms similar or even identical to those described above for the vagina [35].

3. Clitoris

The most recent anatomical description from cadaveric dissections [205] is of a triplanar complex of erectile tissue with a midline shaft lying in the media sagittal approximately 2-4 cm long and 1-2 cm wide, which bifurcates internally into paired curved crura 5-9 cm long. Posterior to the shaft, on either side of the urethra are two separate vestibular bulbs (3-7 cm long of crescentric or triangular shape thought to be spongiosus tissue). The shaft is composed of two chambers, the corpora cavernosa, surrounded by a fibrous sheath (tunica albuginea). It is capped externally by the glans some 2-3mm long and wide consisting of cavernous spongiosus tissue coming from the vestibular bulbs [206] and is normally covered by a protective hood of skin formed from the fusion of the two labia minora. Its parasympathetic innervation comes from the lumbosacral segments L2-S2 while its sympathetic supply is from the hypogastric superior plexus. The pudendal and hypogastric nerves serve its sensory innervation. Sensory nerve terminations include Merkel tactile discs (touch), Pacinian (deep pressure ?) and genital nerve corpuscles, and free nerve endings (pain ?) [34, 207]. There was wide variation in the quantity, quality and location of the various nerve endings.

The clitoris also responds with increased blood flow and tumescence on being stimulated or through sexual arousal. Nitric oxide synthase (NOS), together with many neuropeptides, have been identified in the complex network of nerves in the clitoral tissue by imunohistochemical studies over the last few years. The list includes VIP, PHM (peptide histidine methionine), NPY, CPON (C-flanking peptide of NPY), CGRP (calcitonin gene-related peptide) and substance P [208-210]. The presence of the enzyme NOS in the nerves supplying the clitoral cavernous tissues and in the endothelial cells lining the cavernosus tissue indicates that its vasodilating product NO is involved in the enhanced flow and tumescence while the presence of the vasoconstrictors NPY/CPON suggests that they could throttle venous drainage and facilitate the organ's engorgement. VIP/PHM are known vasodilating neurotransmitters. The exact functions of the other neuropeptides are more problematic. It has been suggested that Substance P, CGRP, and even NO may have sensory roles or may be involved in influencing capillary permeability [210].

4. Uterus

The uterus was said to increase significantly in size during sexual arousal [14] when monitored by palpation, though limited MRI imaging has not confirmed this [28]. It may be that the latter needs better resolution. Odd contractions of the uterus can occur during arousal, but at orgasm a specific pattern of contractions occurs.

D. PSYCHOLOGICAL/ CULTURAL ASPECTS OF WOMEN'S ORGASM

I. PSYCHOSOCIAL FACTORS RELATED TO WOMEN'S ORGASM

The psychosocial factors most commonly discussed in relation to female orgasmic ability include age, education, social class, religion, personality, and relationship issues. While no significant relation between education level and orgasmic ability with a

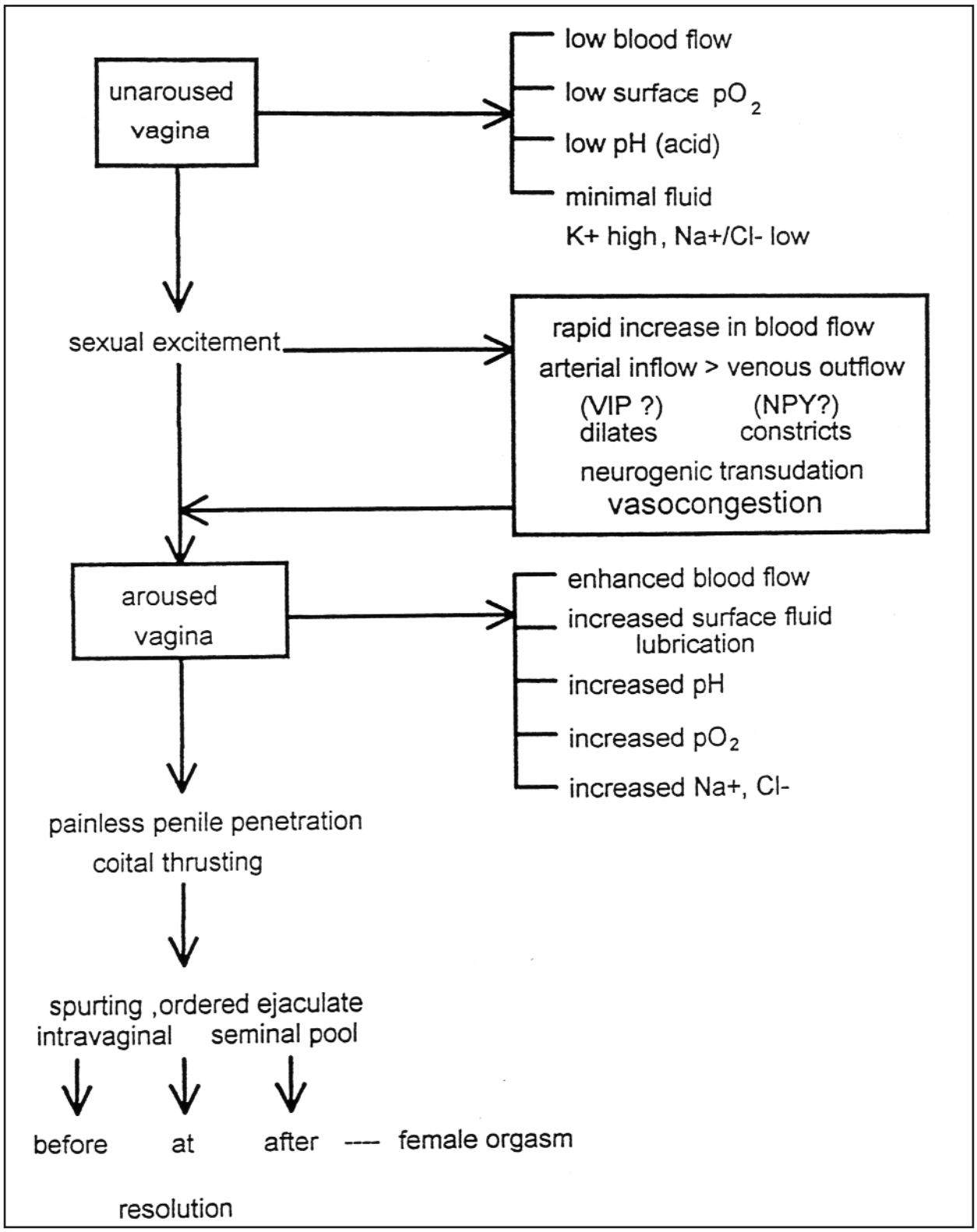

Figure 4 : Summary diagram of the various changes that take place in the human vagina during coitus with and without orgasm.

partner, substantial differences between education level and ability to attain orgasm during masturbation have been reported. Approximately 87% of women with an advanced degree reported "always" or "usually" attaining orgasm during masturbation compared with 42% of women with a high school education

A negative relation between orgasmic ability and high religiosity has been reported. Laumann et al. [211] reported a substantially higher proportion (79%) of women with no religious affiliation reported being orgasmic during masturbation compared with religious groups (53% - 67%). However, there were substantial differences in education levels between religious categories. A relation between improved orgasmic ability and decreased sexual guilt has also been reported [212]. Low orgasmic experience has been consistently related to childhood loss or separation from the father, fathers who had been emotionally unavailable, or fathers with whom the women did not have a positive childhood relationship. Reports of an association between early abuse and anorgasmia are inconsistent (e.g., [213-215]).

Orgasm consistency, quality, and satisfaction in women have been related to relationship factors such as marital satisfaction, marital adjustment, happiness, and stability (for review, see [4]) however rates of orgasm consistency in women are higher during masturbation than with a partner [211]. In summary, there are no consistent, empirical findings that psychosocial factors alone differentiate orgasmic from anorgasmic women. Research that systematically examines these factors among women who are more carefully diagnosed as either meeting or not meeting clinical criteria for Female Orgasmic Disorder is needed.

II. ORGASM AS A GOAL OF WOMEN'S SEXUAL ENCOUNTERS

Perceived wisdom or a sex role stereotype is that men are goal-orientated to achieve orgasm ; if it doesn't occur in a sexual encounter, then they are supposedly dissatisfied and frustrated. For women, it is equally often stated that orgasm is not as highly prized as a goal in such encounters [216]. While some women consider coitus without achieving orgasm unfulfilling and frustrating, especially in relation to the lack of dissipation of their pelvic congestion, others have a high regard for coitus and its pleasures but pay low regard to orgasm per se [7, 217]. Women have been noted to appreciate the "afterglow" of sexual arousal and the body intimacy of being cuddled [218-219] as much as the orgasm itself.

In questionnaires administered to a self-selected rather than a statistically random population, it was reported that women, whether they experienced orgasm or not, gave affection, intimacy and love as major reasons for liking sexual intercourse, and their favorite experience was the act of penetration rather than orgasm itself [32]. All these studies and their conclusions were conducted over 25-40 years ago in America and sexual perceptions of societies and their individuals can change. Waterman & Chiauzzi [220] investigated the relationship between sexual enjoyment and orgasm in couples attending university. They reported "nothing in their data supports the cultural stereotype that orgasms are more important to men than to women". A recent, statistically valid, national survey of British sexual behavior published in 1994 [221] asked both men and women to agree or disagree with the statement "Sex without orgasm cannot be really satisfying". Although nearly half of all men (48.7%) agreed or strongly agreed that orgasm is necessary to male sexual satisfaction, a close 43.3% of all women also concurred.

In women, acute pelvic vasocongestion created by sexual arousal is extensive as it includes uterine, vaginal, clitoral, urethral and labial tissues, pelvic ligaments and possibly even the fallopian tubes [14, 55]. While it is dissipated by orgasm, the dissipation is not normally as rapid or as complete as in the male, and can even be only partial, needing a number of orgasms to occur before its complete resolution to the basal state. In the Masters & Johnson [14] laboratory study of human sexual female responses, the initial recruiting was from prostitutes (p. 10) who suffered from gross varicosities in the genital/pelvic region. The explanation was that their constant sexual arousal during their working day created chronic pelvic vasocongestion that was not dissipated by orgasm (pp. 119-122). Studies of the condition in prostitutes showed that it was accompanied by feelings of pelvic fullness, pressure, cramping, pain, low backache, irritability and sleeplessness, but relief could be instigated by self-induced orgasm. Prostitutes are of course extreme examples but they are illustrative of the condition. Chronic pelvic vasocongestion is an old, established condition first described and examined in the 1940's [55, p. 49; 222-224]. Duncan & Taylor [225] undertook experiments to measure vaginal blood flow and showed that it

increased when subjects were made anxious, depressed and resentful. More recent laboratory studies have reported that female subjects viewing sexually arousing visual stimuli increase their vaginal "blood flow/congestion" as monitored by photoplethysmography without perception of it occurring, even if they subjectively report that they were not consciously sexually aroused or did not enjoy it [226]. The female genital sexual haemodynamic changes of hyperaemia and congestion thus are almost reflex responses even with a claimed negative central sexual arousal. Evidence that female genital sexual arousal is a reflex response is also provided by studies of women with complete SCI who demonstrate reflex genital vasocongestion [227-228]. While no studies have been published comparing the extent of pelvic varicosities in sexually active orgasmic and non-orgasmic women, a "prophylactic" role in their prevention can be envisaged for the female orgasm.

III. CULTURAL ASPECTS OF WOMEN'S ORGASM

Sexual arousal to orgasm through coitus is often thought of as a natural biological act, especially if linked to reproduction. A core concept of social constructionists however is that sexual behavior and identity are learned rather than intrinsic, and culture with its social and historical factors plays a large role in shaping, or at least trying to shape, an individual's sexuality [229]. A very obvious involvement of culture/society on female sexuality has been the acknowledgement of female orgasms, which in reality means the acceptance of female sexual pleasure. Anthropologists have noted that in cultures that expect women to enjoy sex as men do the women have orgasms, while in those cultures that censor such a concept women have more difficulty attaining orgasm. Instances of societies that foster sexual pleasure for women and expect them to enjoy coitus include the Mundugumor [230] and the Mangaia [231]. Mangaian women are taught to have orgasms, hopefully two or three to her male partner's one and to try to attain mutual orgasm. Mangaian males who are not able to give their partners multiple orgasms are not held in high esteem. At the other end of the spectrum are societies that assume women will have no pleasure from coitus and that the female orgasm does not exist. The Arapesh [232] are such a society, as they do not even have a word in their language for the female orgasm. In a similar vein, the Sambia people of the Highlands of New Guinea [233] accord the clitoris (lakandiku) no function or importance and it is never mentioned in public by men. Moreover men deny that there is a female orgasm (imbimboogu).

E. FEMALE ORGASMIC DISORDER

I. INTRODUCTION

Findings from the National Social and Health Life Survey conducted in the early 1990s [211] suggest that orgasmic problems are the second most frequently reported sexual problems in women. In this random sample of 1,749 US women, 24% reported a lack of orgasm in the past year for at least several months or more. This percentage is comparable to clinic-based data. Rosen et al. [234] noted 29% of 329 healthy women (ages 18-73) who attended an outpatient gynecological clinic reported orgasmic problems, and Read et al. [235] reported 23% of 104 women (18-65+) attending a U.K. general practice clinic reported anorgasmia. A precise estimate of the incidence of orgasmic disorder in women is, however, difficult to determine because few well-controlled studies have been conducted, and definitions of orgasmic disorder vary widely between studies depending on the diagnostic criteria used. The DSM-IV-TR [236], defines Female Orgasmic Disorder (302.73) using the following diagnostic criteria.

Persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase. Women exhibit wide variability in the type or intensity of stimulation that triggers orgasm. The diagnosis of Female Orgasmic Disorder should be based on the clinician's judgment that the woman's orgasmic capacity is less than would be reasonable for her age, sexual experience, and the adequacy of sexual stimulation she receives.

The DSM-IV-TR uses the terms lifelong versus acquired and generalized versus situational. However, some studies of orgasm in women use the term "secondary" with little clarity as to whether this is an acquired inability to orgasm under any circumstances or is in fact a situational disorder possible acquired rather than lifelong. The International Statistical Classification of Diseases and Related Health

Problems (ICD-10) defines Orgasmic dysfunction (F52.3) simply as "Orgasm either does not occur or is markedly delayed". Regarding women who can obtain orgasm during intercourse with manual stimulation but not intercourse alone, the clinical consensus is that she would not meet criteria for clinical diagnosis.

II. ORGASM IN MENOPAUSE

A critical period in the ageing process for women is the premenopausal-postmenopausal change. Compared to young women, the response to sexual stimulation in the laboratory in postmenopausal women showed delay in achieving full tumescence in the clitoris, marked decrease in breast volume engorgement, no engorgement of the uterus, delayed or absent vaginal lubrication and decreased vaginal expansion. At orgasm, there were fewer vaginal contractions and rarely any rectal ones. The reduced number of vaginal and anal contractions, possible indicators of the intensity of pleasure according to Masters & Johnson [14], suggested a "generalized reduction in the intensity of orgasm expression". Unfortunately, their wording is ambiguous and could mean either a real decrease in the intensity of orgasm or a decrease in the physical expression of orgasm at various sites. This decrease in intensity of orgasm was also reported by Basson [237] in androgen deficient menopausal women. These women also had difficulty in trying to focus during arousal to orgasm.

In some menopausal women, pain can occur during and after the uterine/vaginal contractions of orgasm. Levin [194] suggested that in the premenopausal women, contractions of the vagina and uterus are induced by a neurotransmitter that has to overcome the inhibitory action of any released VIP. In the menopausal state, however, VIP is probably ineffective in relaxing smooth muscle [238] so that the contractions of the uterus/vagina induced by the neurotransmitter at orgasm is unopposed, leading to spasmodic type contractions creating anoxia and thus pain. Giving oestrogen and progesterone together causes relief, but neither is adequate separately [14].

III. TREATMENT

The treatment of anorgasmia has been approached from psychoanalytic, cognitive-behavioral, pharmacological, and systems theory perspectives [239]. Substantial empirical outcome research is available only for cognitive behavioral and, to a lesser degree, pharmacological approaches. Hence, this section will provide a review only of cognitive behavioral techniques and pharmacotherapy used to treat female anorgasmia. To this end, ***Tables 6, 7, and 8*** provide a summary of controlled and uncontrolled studies by treatment techniques. Definitive recommendations for treatment are based solely on controlled outcome research. One of the difficulties in assessing treatment effectiveness for anorgasmia is the nebulous manner in which studies often define orgasmic dysfunction. While some studies use clinician interviews to determine whether women meet criteria for primary or secondary anorgasmia, others rely solely on participant verbal reports of orgasmic difficulty or the results of brief self-report inventories. For this reason, where possible, information on the way in which orgasmic dysfunction is defined is included in the Tables.

1. Cognitive-behavioral approaches

Cognitive-behavioral therapy for anorgasmia focuses on promoting changes in attitudes and sexually-relevant thoughts, decreasing anxiety, and increasing orgasmic ability and satisfaction. Behavioral exercises traditionally prescribed to induce these changes include directed masturbation, sensate focus, and systematic desensitization. Sex education, communication skills training, and Kegel exercises are also often included in cognitive-behavioral treatment programs for anorgasmia.

a) Directed masturbation

Given masturbation can be performed alone, any anxiety that may be associated with partner evaluation is necessarily eliminated. Related, the amount and intensity of sexual stimulation is directly under the woman's control and therefore the woman is not reliant upon her partner's knowledge or her ability to communicate her needs to her partner. Research that shows a relation between masturbation and orgasmic ability provides empirical support for this treatment approach [211].

Directed masturbation (DM) has been used to effectively treat anorgasmia in a variety of treatment modalities including group, individual, couples therapy, and bibliotherapy. As can be seen in ***Table 6***, a number of outcome studies and case series report directed masturbation is highly successful for treating primary anorgasmia. In a controlled comparison of therapist-directed group masturbation training,

Table 6 . Psychological Treatment of Orgasmic Dysfunction

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
DIRECTED MASTURBATION					
Controlled Outcome Studies					
Heinrich (1976) [240]	44	*M* age=25; 20 married, 24 with regular partner	Primary anorgasmia	DM (G) vs. DM bibliotherapy (I) vs. WL; DM: 10 sessions/5 wk; DM bibliotherapy: 1 session	2 mo: DM: 100% orgasmic with masturbation (om), 47% coitally orgasmic (co); DM bibliotherapy: 47% om, 13% co; WL: 21% om, 0% co
Munjack et al. (1976) [247]	22	12 prim, 10 sec	Primary and secondary anorgasmia	SD, DM, assertiveness training, modeling, sexual edu (I/C) vs. WL; 22 weekly sessions	Tx > WL orgasmic ability; no difference between prim and sec
Riley & Riley (1978) [248]	SF (n=15) DM + SF (n=20)	*M* age=26; married	Primary anorgasmia, defined as orgasmic inability regardless of type of sexual stimulation	DM and SF (C) vs. SF (C); 6 weekly and 6 bi-monthly sessions	DM and SF: 18/20 orgasmic; SF: 8/15 orgasmic; 1 yr follow-up: gains maintained
McMullen & Rosen (1979) [241]	DM Bibliotherapy (n=20) DM Instructional (n=20) WL (n=20)	*M* age=29; 30 married, 30 single	Primary anorgasmia, defined as the orgasmic inability through any means of sexual stimulation; assessed via clinician interview, self-report, General Information Questionnaire and Sexual Behavior Inventory	DM Bibliotherapy (I) vs. DM Instructional videotape (I) vs. WL; 6 sessions/6 wk	Bibliotherapy: 65% orgasm with masturbation (om), 50% coitally orgasmic (co); Instructional: 55% om, 30% co; WL: 0% om, 0% co; 1 yr follow-up: gains maintained/improved

Table 6. Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Reisinger (1979) [249]	3	*M* age =33; married 8-15 yrs	Secondary anorgasmia, coitally and through masturbation	DM with erotic video 8-13 sessions; stimulation by partner w/o training 2-6 sessions; stimulation by partner w/training 6-10 sessions; solitary stimulation w/o erotic aids 2-3 sessions; stimulation w/partner 4-7 sessions	DM: 3/3 orgasmic ability through masturbation; limited orgasmic success w/o partner training; 67% orgasmic ability with partner training; 2, 6 mo follow-ups: 80% orgasmic ability with and without partner stimulation
Andersen (1981) [250]	30	*M* age=25; 25 married, all with regular partners; some sexual aversion	Self-reported primary anorgasmia, also assessed via Sexual Interaction Inventory	SD (G) vs. DM (G) vs. WL; 10 sessions/5 wk	DM > SD, WL on orgasmic response; 6 wk follow-up: DM > SD on orgasmic response
Delehanty (1982) [251]	28	*M* age =30	Preorgasmic: no history of orgasm within previous 5 yrs or primary anorgasmia; assessed via self-report and orgasm checklist	DM and assertiveness training in group co-therapy format for 10 wk vs. WL	82% orgasmic success with tx
Heiman & LoPiccolo (1983) [252]	41	*M* age=30; 25 prim, 16 sec, absence of severe marital distress	Primary and secondary anorgasmia	CBT, communication training, DM, SF, systems conceptualization (C) vs. WL; 15/1-hr sessions	Prim and Sec: Increased duration foreplay and si; Prim: increased frequency si, increased orgasmic response during masturbation and si; Sec: increased orgasmic response during si, increased initiation of sexual activity; 3 mo follow-up: Prim: gains maintained, Sec: orgasmic gains maintained, decreased duration foreplay and si

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Bogat, Hamernik, & Brooks (1987) [253]	11	N/A	Self-reported preorgasmic (less than 10% of time) with desire to improve ability, also assessed with Women's Orgasmic Efficacy and Comfort Scale	DM vs. no treatment (C); 10 sessions	80% Improvement in orgasmic success in tx vs. controls
Eichel, Eichel, & Kule (1988) [254]	CAT (n=22) Control (n=43 men and women)	CAT: *M* age =40; Control: *M* age = 39; interest in sexual enhancement	Orgasmic function assessed via orgasmic attainment criteria scale	Coital Alignment technique (CAT) (C) vs. no treatment (C)	CAT group: improvement in frequency of orgasm, simultaneous orgasm, & orgasm satisfaction compared to controls; use of CAT by both groups correlated with improved frequency of all orgasm variables
Hurlbert & Apt (1995) [242]	CAT (n=19) DM (n=17)	*M* age=28; 36 sec; *M* yr married = 5	Secondary anorgasmia, assessed via self-report and sex diary	Coital alignment technique (CAT) I vs. DM I ; 4, 30-min sessions involving assertiveness training, communication skills, and SF plus 4, 10-min telephone contacts	CAT: 37% substantially improved, 58% moderately improved orgasmic ability during si; DM: 18% substantially improved, 35% moderately improved orgasmic ability during si
No Control Outcome Studies					
LoPiccolo & Lobitz (1972) [255]	8	Married	Primary anorgasmia, assessment method not specified	DM (I) modeled after Masters & Johnson; Kegel exercises; 15 sessions	8/8 Orgasmic with masturbation, 6/8 coitally orgasmic; 6 mo follow-up: gains maintained
Lobitz & LoPiccolo (1972) [256]	13	Married	Primary anorgasmia	DM (I with partner participation); 15 sessions	13/13 Orgasmic with masturbation, 13/13 coitally orgasmic 50% of time

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Barbach (1974) [257]	83	19-48 yo	Primary anorgasmia, defined as no orgasmic experience	DM (G); 10 sessions/5 wk	92% Orgasmic with masturbation
Wallace & Barbach (1974) [258]	17 (of 83 in Barbach, 1974)	*M* age =28; 11/17 married; all with partners	Primary anorgasmia	DM (G); 10 sessions/5 wk	100% Orgasmic with masturbation 87% orgasmic with partner; 8 mo follow-up: gains maintained
McGovern, Stewart, & LoPiccolo (1975) [259]	12	6 prim, 6 sec	Primary anorgasmia and secondary anorgasmia	Sexual and communication skills training, anxiety re-education, DM; 15 sessions	Prim: 6/6 Increased orgasmic ability; Sec: no change in orgasmic ability
Schneidman & McGuire (1976) [260]	20	10 < 35 yo, 10 > 35 yo; 70% prim; some problems with male ejaculatory control	Primary anorgasmia (except nocturnal orgasms while dreaming)	Variation of Masters & Johnson (sexual edu, group discussions, DM, couples tx) I; 10 wk	< 35 yo: 70% orgasmic during masturbation, 0/10 coitally orgasmic; > 30 yo: 40% orgasmic during masturbation, 0/10 coitally orgasmic; 6 mo: < 35 yo: 80% orgasmic during masturbation, none orgasmic during si; > 30 yo: 60% orgasmic during masturbation, 1/10 orgasmic during si
Kirkpatrick et al. (1977) [261]	4	Not available.	N/A	DM (G)	4/4 orgasmic during masturbation, 75% coitally orgasmic
Leiblum & Ersner-Hershfield (1977) [262]	16	23-43 yo; 12 married	Primary and secondary anorgasmia and general orgasm dysfunction, assessed via General Information Questionnaire	DM, sensate focus, and sexual edu (G); 8 - 10sessions/5-8 wk	88% orgasm with masturbation

Table 6. Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Sotile, Kilmann, & Follingstad (1977) [263]	6	3 prim, 3 sec; 15 previous SD sessions	Primary and secondary anorgasmia, assessed via Sexual Interaction Inventory	Sexual and communication skill training, sexual edu, SF, DM, Kegel ex, role-play orgasm (C); 6/1 _-hr sessions	No change in orgasm
Ersner-Hershfield & Kopel (1979) [264]	22	*M* age=26; 14/22 married; 13 prim, 9 sec	Preorgasmic, defined as either no orgasmic experience or less than 10% success achieving orgasm in the past, assessed via Survey of Sexual Activities	DM: Spaced vs. massed sessions (G,I) vs. DM: spaced vs. massed sessions (G,C), all fashioned after Barbach (1975) and Heiman, LoPiccolo, & LoPiccolo (1976) formats; 10 sessions/5 wk	91% Orgasmic with masturbation, 73% orgasmic with partner; no difference GI vs. GC or spaced vs. massed sessions; 10 wk follow-up: 82% orgasmic with partner
Barbach & Flaherty (1980) [265]	26	19 to 60 yo; follow-up on previous study	Secondary anorgasmia	DM, communication training (I); 10, 1 _ hr sessions	1-2 yr follow-up: 60% increased orgasmic frequency with partners
Kuriansky, Sharpe, & O'Connor (1982) [266]	19	*M* age=30; previous or current psychotherapy; 3/19 situationally orgasmic	Primary or secondary anorgasmia assessed via self-report and clinician interview; Orgasm Hierarchy Scale	SD, DM, assertiveness training (G,I), based on Barbach (1974), Lobitz & LoPiccolo (1972), and LoPiccolo & Lobitz (1972); 10 sessions/5 wk	18/19 Orgasmic; 68% orgasmic via self-stimulation; 21% orgasmic with partner; 2 yr follow-up: 16/19 orgasmic, 37% orgasmic via self-stimulation, 47% orgasmic with partner
Adkins & Jehu, (1985) [267]	6	*M* age =28; M yr in relationship = 3.5	Self-reported primary orgiastic dysfunction	DM and bibliotherapy; 10 sessions/10 wk	3/6 Orgasmic success at partner-involvement with no intercourse phase; authors suggest use of vibrators aided orgasm success; 6 mo follow-up: 3/6 orgasmic success via masturbation or coitus with clitoral stimulation

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
De Amicis et al. (1985) [268]	22	*M* age=34; *M* yr married=13; 13 prim, 9 sec	Primary and secondary anorgasmia	Sensual awareness, SF, DM, communication training, modification of sexual interactions (C); 15-20 sessions	No change in orgasmic ability, increased sexual satisfaction; 3 yr follow-up: Prim: increase in orgasmic ability with genital caress; Sec: increase in orgasmic ability during masturbation
Wakefield (1987) [269]	15	Reanalysis of data from Ersner-Hershfield & Kopel (1979)	Self-reported primary anorgasmia, assessed via Survey of Sexual Activities	DM: spaced vs. massed sessions (GI) vs. DM: spaced vs. massed sessions (GC); 10 sessions/5 wk	80% orgasm via masturbation; 7% orgasm via partner stimulation; no coital orgasm; 10 wk follow-up: 93% orgasm via masturbation, 20% orgasm via partner stimulation; no coital orgasm
Kaplan (1992) [270]	21	Sexuality seminar participants; 21/30 attempted CAT	N/A	Coital alignment technique (CAT) for 1-3 sexual encounters/2 wk	1/21 Enhanced coital orgasm; 20/21 no enhanced or increased coital orgasm, nor increased simultaneous orgasm
SYSTEMATIC DESENSITIZATION					
Controlled Outcome Studies					
Husted (1972; 1975) [271-272]	30	Mixed sexual dysfunction; all with partners; sexual anxiety	N/A	SD: Imaginal (I) vs. (C); vs. *in vivo* (I) vs. (C) vs. No-treatment control; Imaginal *M*=8 sessions, *in vivo M*=13 sessions	SD: Decreased anxiety, increased coital frequency and orgasmic ability with masturbation; no difference (I) vs. (C) or imaginal vs. *in vivo*
Obler (1973) [273]	37	Mixed sexual dysfunction; marital status matched across groups	N/A	SD with videotapes (I) vs. Psychoanalytic tx with videotapes (G) vs. WL; SD: 15 45-min sessions; Psychoanalytic: 10 75-min sessions	SD: 85% orgasmic; Psychoanalytic: 36% orgasmic WL: 23% orgasmic SD> Psychoanalytic, WL on decreased anxiety

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Mathews et al. (1976) [274]	18	*M* age=28; 13 prim, 5 sec; 17/18 low sexual desire/arousal	Primary and secondary anorgasmia	SD, sexual tx (C) vs. SF, sexual tx (C) vs. SF, bibliotherapy (C); 10 sessions; 3 sessions and 10 wk mailing for SF, bibliotherapy	2/18 Increased orgasmic ability; no difference between groups; 4 mo follow-up: no difference between groups
Wincze & Caird (1976) [275]	21	18-38 yo; 16 prim, 5 sec; 19/21 married; sexual anxiety	Frigidity, including "essential" sexual dysfunction	SD Imaginal (I) vs. SD video (I) vs. WL; *M*= 10 sessions/ 2-7 wk	SD: 40% orgasmic; no difference between imaginal/video groups; 1-3 mo follow-up: 25% orgasmic ability
Nemetz, Craig, & Reith (1978) [276]	SD (I) (n=8) SD (G) (n=8) Control (n=6)	21-39 yo; 7 prim, 15 sec; sexual anxiety; all with regular partners	Primary and secondary anorgasmia	SD (I) vs. SD (G) vs. Control; 5 sessions/3 wk	No difference between groups in orgasm; 3 wk, 1yr follow-up: gains maintained
O'Gorman (1978) [277]	40	*M* age=36; low sexual desire/arousal, some dysparuenia/Vaginismus	Frigidity, including orgasm dysfunction	SD, sex edu (G), partner-only discussion groups vs. SD, intravenous methoxitone sodium to induce relaxation (I with partner participation); SD (G) 20 1-hr sessions; SD (I) 15 10-min sessions/ 10 wk	SD, sex edu (G): 63% successful; SD, methoxitone sodium (I): 37% successful
Andersen (1981) [250]	30	*M* age=25; 25 married, all with regular partners; some sexual aversion	Self-reported primary anorgasmia, also assessed via Sexual Interaction Inventory	SD (G) vs. DM (G) vs. WL; 10 sessions/5 wk	DM > SD, WL on orgasmic response; 6 wk follow-up: DM > SD on orgasmic response

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Obler (1982) [278]	Integrated (n=8) Couples (n=8) No treatment (n=10)	18-36 yo; married or cohabiting for over 2 yrs; no previous psychotherapy	N/A	42 wk of Integrated hypnoanalytic/behavioral group vs. 16 wk of Cotherapist/Couples vs. No tx; 1 yr	Integrated: 7/8 self-reported orgasmic ability over 60% of time; Cotherapist/ Couples: 2/8 self-reported orgasmic ability over 60% of time; No tx: no self-reported orgasmic ability
Fichten, Libman, & Brender (1983) [279]	23	*M* age=33; sec; *M* yr married = 10; Orgasmic < 25% of time	Secondary anorgasmia, defined as at least 1 orgasmic experience, dissatisfaction with orgasmic frequency, and narrow range of orgasmic stimulation	Sexual information, relaxation, Kegel ex, DM, SF, sexual communication training, ban on si: (C) vs. (G) vs. minimal contact bibliotherapy; 14 wk	No change in orgasm
Own Control or Wait-list Controlled Outcome Studies					
Munjack et al. (1976) [247]	22	*M* age =29; *M* age married=67; 12 prim, 10 sec; no women had experienced orgasm within 1 yr	Primary anorgasmia (except orgasm in sleep) or secondary anorgasmia	SD, DM, assertiveness training, modeling, sexual education (I/C) vs. WL; 22 weekly sessions	Tx > WL orgasmic ability; no difference prim vs. sec
Sotile & Kilmann (1978) [280]	22	*M* age =28; 8 prim, 14 sec; all with partners; no partner sexual dysfunction; sexual anxiety	Primary and secondary anorgasmia, assessed via Sexual Behavior and Attitudes Questionnaire and self-report	Sexual education followed by SD (G) or WL for 16 sessions/8 wk	Increased noncoital orgasmic frequency; sec > prim orgasmic frequency following tx; 6 wk follow-up: gains maintained

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
No Control Outcome Studies					
Cooper (1970) [281]	50	N/A	Coitally anorgasmic, assessed by clinical interview	*In vivo* SD, sex education, psychotherapy (I); 21 sessions/1 yr	24/50 coitally orgasmic; 26/50 unchanged or worse
Jones & Park (1972) [282]	55	Anxiety; sexual shame	Primary anorgasmia	SD with Brevital injections to induce relaxation (I with partner participation); *M*=14 sessions	82% orgasmic success
SENSATE FOCUS / OTHER					
Controlled Outcome Studies					
Mathews et al. (1976) [274]	18	*M* age=28; 13 prim, 5 sec; 17/18 low sexual desire/arousal	Orgasmic dysfunction, defined as failure to experience orgasm and assessed via clinician interview	SD, sexual tx (C) vs. SF, sexual tx (C) vs. SF, bibliotherapy (C); 10 sessions; 3 sessions and 10 wk mailing for SF, bibliotherapy	2/18 Increased orgasmic ability; no difference between groups; 4 mo follow-up: no difference between groups
Carney, Bancroft, & Mathews (1978) [283]	Testosterone (n=16) Diazepam (n=16)	*M* age =29; sexual anxiety; Vaginismus or orgasm dysfunction as primary complaint excluded	Secondary anorgasmia assessed via self-report, clinician rating and independent assessor rating	SF weekly: testosterone, 10 mg daily (T) vs. diazepam, 10 mg daily (C) vs. SF monthly: T vs. diazepam (C); SF weekly: 16 sessions, SF monthly: 5 sessions	No difference in orgasm between weekly vs. monthly T > diazepam frequency of orgasm; 6 mo follow-up (after drug discontinuation): gains maintained

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Roughan & Kunst (1981) [284]	PC Group (n=14) Relaxation (n=12) Control (n=14)	*M* age =32; 14 participants met criteria for orgasmic dysfunction	Primary anorgasmia or secondary anorgasmia lasting over 2 yrs	PC (G): PC muscle exercises for 50 contractions, 5x daily for 12 wk vs. Relaxation (G): physical relaxation for 12 wk vs. no tx	No relationship between PC muscle tone and orgasmic ability in any group
Fichten, Libman, & Brender (1983) [279]	23	*M* age=33; *M* yr married = 10	Secondary anorgasmia	Sexual information, relaxation, Kegel ex, DM, SF, sexual communication training, ban on si: (C) vs. (G) vs. minimal contact bibliotherapy; 14 wk	SF: No change in orgasm; increase in enjoyment of noncoital sexual caressing and si
Chambless et al. (1984) [285]	16 (group n's not specified)	*M* age =27	< 30% orgasm success through coitus; assessed with Women's Sexuality Questionnaire	Kegel ex vs. Attn. placebo (nonsexual imagery) vs. WL; 6 wk	No differences in coital orgasmic frequency despite improvement in each group; no change in perceived vaginal stimulation during orgasm in any group
LoPiccolo et al. (1985) [286]	31	*M* age =35; 12 prim, 19 sec; *M* yr married=13	Primary and secondary anorgasmia	CBT sexual therapy (LoPiccolo & Hogan, 1979) vs. WL (C), both for 15 1-hr sessions	Prim and sec: Increase in orgasm with masturbation; 3 mo follow-up: gains maintained/improved
Kilmann et al (1986) [245]	55	*M* age =33; 51 married; all with partners; no Dyspareunia or Vaginismus, no premature ejaculation in partners	Secondary anorgasmia for 5 mo through si or clitoral stimulation and dissatisfied with coital orgasmic ability; assessed via clinician interview and Sexual Behavior and Attitudes Questionnaire	2 2-hr sessions sex education followed by Communication skills (C/G) vs. Sexual skills (C/G) vs. WL vs. Attn-placebo	Communication and sexual skills > controls in coital orgasm ability; no difference between groups; 6 mo follow-up: gains decreased, no difference between groups

Table 6 . sychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Morokoff & LoPiccolo (1986) [287]	43	*M* age =30; prim; *M* yr married=9; no male sexual dysfunction, no psychosis or depression	Primary anorgasmia, assessed via Sexual History Form	DM and Bibliotherapy in either Minimal therapist contact for 4 sessions (MTC; n=14) vs. Full therapist contact for 15 sessions (FTC; n=29)	Increased orgasmic ability with masturbation and si; MTC > FTC on increased frequency orgasm with masturbation
Kilmann et al. (1987) [288]	11	*M* age =30; 10 married; no premature ejaculation in partners	Secondary anorgasmia, defined as 50% coital orgasmic success or less over 5 mo and dissatisfaction with orgasmic frequency, assessed via structured interviews, Sexual Interaction Inventory and Sexual Behavior and Attitudes Questionnaire	2 2-hr sessions sex education followed by communication and sexual skills vs. WL vs. Attn-placebo	Tx > WL, Attn-placebo: increase in orgasmic ability with tx
Milan, Kilmann, & Boland (1988) [289]	38	*M* age =33; sec; *M* yr relationship=10; regular sexual partners with no sexual dysfunction; 9% orgasmic frequency pretreatment	Secondary anorgasmia, assessed via scale adapted from the Sexual Behavior and Attitudes Questionnaire	10 2-hr sessions/5 wk of sex education plus either: communication skills vs. sexual skills vs. condensed sex and communication skills vs. didactic lecture vs. WL	2-6 yr: No difference between tx groups, WL on sexual or relationship functioning

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Van Lankveld, Everaerd, & Grotjohann (2001) [290]	Bibliotherapy (n=9) WL control (n=9)	*M* age =37; *M* sexual dysfunction duration= 8 y; Hyposexual Desire Disorder; Vaginismus; Dyspareunia	DSM-IV diagnosis of orgasmic dysfunction via structured interview, with no distinction between primary and secondary anorgasmia; assessed via self-report and Golombok Rust Inventory of Sexual Satisfaction	Bibliotherapy (including communication skills, sexual education, and SF) and CBT with telephone support vs. WL; 10 wk	No improvement in orgasm in tx vs. controls
No Control Outcome Studies					
Lazarus (1963) [291]	16	*M* age =25; married; some decreased desire/arousal	Persistent orgasmic dysfunction, method of assessment not specified	SD (I), *M*=29 sessions over 6 mo	9/16 “nearly always achieve orgasm”; 15 mo follow-up (4 patients): gains maintained or improved
Masters & Johnson (1970) [243]	342 (1959-1964)	193 prim; 11 masturbatory dys; 106 coital dys; 32 random	Primary and secondary anorgasmia	Sex education, SF, communication training, *in vivo* SD (C); 14 sessions/daily	Prim: 83% orgasmic; Masturbatory: 91% orgasmic; Coital: 80% orgasmic; Random: 63% orgasmic; 5 yr follow-up: Prim 1% relapse; Sec 2% relapse
Blakeney et al. (1976) [292]	38	10 prim, 28 sec; some male sexual dysfunctions	Primary and secondary anorgasmia	4-hr interview and 2 _ day workshops based on Masters & Johnson (C)	Prim: 70% orgasmic; Sec: 57% orgasmic
Sotile, Kilmann, & Follingstad (1977) [263]	6	3 prim, 3 sec	Primary and secondary anorgasmia	Sexual and communication skill training, sexual education, SF, DM, Kegel ex, role-play orgasm (C); 6/1 _-hr sessions	Decreased sexual anxiety, increased sexual communication

Table 6. Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Golden et al. (1978) [293]	17	*M* age =27; 14/17 married	Secondary anorgasmia as primary diagnosis, assessed via Goals for Sexual Therapy: Female form	Couples assigned SF, sexual skills, and communication skills in either a tx (G) vs. tx (C) format; 12 wk	Couple and group tx improved orgasm satisfaction (data suggests group tx slightly more beneficial)
Jankovich & Miller (1978) [294]	17	19 – 38 yo	Primary anorgasmia assessed via interview	Therapy and audiovisual sexual education over a single week (G)	7/17 experienced orgasm within a week: 4 via masturbation, 2 via partner manual stimulation, 1 via manual stimulation and si
Dodge, Glasgow, & O'Neill (1982) [295]	13	*M* age =late 20s	Primary or secondary anorgasmia (orgasmic mainly through masturbation), Sexual Interaction Inventory and Sexual Arousal Inventory	Tx of minimal-contact bibliotherapy in 3, _ h therapy sessions vs. delayed treatment group given information on human sexuality	Tx increased coital orgasm; 2/3 prim attained orgasm with tx vs. 0/2 orgasm in prim controls; no change orgasm via masturbation for tx or control; 6 wk follow-up: increase in coital orgasmic ability with tx
Cotton-Huston & Wheeler (1983) [296]	70	*M* age of treatment group=34; *M* age of control group=31; both groups with orgasmic dysfunction	Primary or secondary anorgasmia, method of assessment not specified	Combination of group and sex therapy approaches vs. no tx	Increased masturbation and orgasm through masturbation in tx vs. controls
Kilmann et al. (1983) [297]	48	*M* age =33; *M* yr married=10; 8.4% coital orgasm frequency; *M* dysfunction persistence of 9.6 yr	Secondary anorgasmia, with orgasm frequency less than 50% for 5 mo, assessed in interview; orgasmic ability also assessed via Sexual Behavior and Attitudes Questionnaire	Sex education during 2, 2-hr sessions within a single wk	Increases in orgasmic frequency subscale; increases in coital, noncoital, and masturbatory orgasm frequency

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Trudel & Saint-Laurent (1983) [298]	PC Group (n=6) Relaxation Group (n=6)	N/A	Orgasmic ability via clitoral stimulation, not coitus (assessed through interview)	PC group: 20 m daily PC exercises for 8 wk; Relaxation group: 20 m daily exercises in sexual awareness, relaxation, and breathing for 8 wk	No differences between groups in coital orgasm ability; in the Relaxation group, 1 orgasmic woman during tx, 1 woman orgasmic post-tx (not attributed to tx effects)
Libman et al. (1984) [299]	Couple (n=7) Group (n= 8) Bibliotherapy (n=8)	*M* age =33; *M* yr married=10; 25% orgasmic frequency	Secondary anorgasmia assessed via interview and Jewish General Hospital Sexual Behavior Questionnaire	15, 1-hr sessions over 14 wk (G) vs. all-female groups for 15, 1.5 hr sessions over 14 wk (G) vs. Minimal Contact Bibliotherapy: 2 sessions at beginning and end of 14 wk period	Therapy (C) and bibliotherapy > therapy (G) in orgasm with manual stimulation; Therapy (C) > other groups in orgasm via giving and receiving manual stimulation ; overall gains in orgasm via masturbation, receiving manual stimulation, giving and receiving manual stimulation, receiving oral stimulation across conditions
De Amicis et al. (1985) [268]	22	*M* age=34; *M* yr married=13; 13 prim, 9 sec; low arousal and desire, Vaginismus, and/ or Dyspareunia	Primary and secondary anorgasmia, assessed with diagnostic criteria of Schover et al. (1982) and Sexual History Form	Sensual awareness, SF, DM, communication training, modification of sexual interactions (C); 15-20 sessions	No change in orgasm; 3 yr follow-up: Prim: increase in orgasm with genital caress; Sec: some increase in orgasm during masturbation
Sarwer & Durlak (1997) [300]	34	Couples ages 20-60 yo; married	DSM-III diagnosis of inhibited orgasm	Behavioral treatment involved SF for 30 min/d, video, lecture, and sex education material over 7 wk of weekly sessions of 4 h	65% resolved orgasm dysfunction by end of tx
Billups et al. (2001) [301]	32	Pre- and post-menopausal women with and without FSD	Orgasm function assessed with Female Intervention Efficacy Index	6 at-home sessions of clitoral vacuum therapy; 5-15 m with or without partner	FSD: 55% Increased orgasm; non-FSD: 42% increased orgasm

Table 6 . Psychological Treatment of Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
McCabe (2001) [302]	36	*M* age =36; low sexual interest; sexual arousal disorder; Vaginismus	Orgasm dysfunction, assessed via Sexual Dysfunction Scale	CBT, SF, interpersonal communication; sexual skills; alleviating sexual anxiety	Anorgasmia decreased from 66.7% to 11.1% post-tx; increase in positive sexual attitudes
Zajecka et al. (2002) [303]	CBASP (n=140) Nefazodone (n=144) Combined (n=155)	*M* age =43; 65% of sample female; depression; 48% women reported baseline sexual dysfunction	Difficult, less intense, or lack of orgasm	Cognitive Behavioral Analysis System of Psychotherapy (CBASP) 2x weekly, nefazodone (200-600 mg/d), or combination for 12 wk period	N.s. improvement in orgasm with CBASP, nefazodone and combination tx groups at 12 wk compared to baseline

Note: SD = systematic desensitization, DM = directed masturbation, SF = sensate focus, CBT = cognitive-behavioral therapy; WL = wait-list, (I) = individual therapy, (C) = couples therapy, (G) = group therapy (GI) = group/individual therapy, (GC) = group/couples therapy, prim = primary orgasmic dysfunction, sec = secondary orgasmic dysfunction, si = sexual intercourse.

Table 7. Pharmacological Treatments for Non-Antidepressant-Induced Orgasmic Dysfunction

References	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
ArginMax					
Ito, Trant, & Polan (2001) [305]	77	*M* age =43; 6 subjects with previous sexual dysfunction	Orgasm function assessed with Female Sexual Functioning Index	ArginMax herbal supplement for 4 wk vs. placebo	47.1% of ArginMax tx improvement in orgasm function at 4 wk vs. 30.2% in placebo group
Bupropion					
Modell, May, & Katholi (2000) [304]	20	21-54 yo; healthy	Self-reported secondary anorgasmia as inability to achieve or delay of orgasm in appropriate time frame; orgasm function also assessed with modified sexual satisfaction questionnaire	3 wk placebo dose, 3 wk bupropion-SR (150 mg) once daily plus placebo dose, 3 wk bupropion-SR (150 mg) 2x daily	No improvement in orgasm, satisfaction, or intensity beyond placebo with either 150 or 300 mg doses
Nefazodone, Psychotherapy					
Zajecka et al. (2002) [303]	Nefazodone (n=144) CBASP (n=140) Combined (n=155)	*M* age =43; 65% of sample female; depression; 48% women reported baseline sexual dysfunction	Difficult, less intense, or lack of orgasm	Nefazodone (200-600 mg/d), Cognitive Behavioral Analysis System of Psychotherapy (CBASP) 2x weekly, or combination for 12 wk period	Improvement in orgasm in nefazodone, CBASP, and combination tx groups at 12 wk vs. baseline

Table 7. Pharmacological Treatments for Non-Antidepressant-Induced Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Definition of Anorgasmia	Treatment	Outcome
Sildenafil					
Kaplan et al. (1999) [161]	12	*M* age =52; postmenopausal	Self-reported orgasmic difficulty or inability via Index of Female Sexual Function	Sildenafil (50 mg) 1 h before sexual activity	At 12 wk, 7.4% improvement in orgasm function
Berman et al. (2001) [162]	44	*M* age =46; all women with Female Sexual Arousal Disorder; hypoactive sexual desire disorder	Difficulty or inability to achieve orgasm, assessed via Brief Index of Sexual Functioning (BISF-W)) and Female Intervention Efficacy Index (FIEI)	Sildenafil (100 mg) at second visit and 6-wk home supply	BISF-W: Increase in orgasm; FIEI: 67% increased orgasmic ability
Testosterone					
Carney, Bancroft, & Mathews (1978) [283]	Testosterone (n=16) Diazepam (n=16)	*M* age =29; sexual anxiety; Vaginismus or orgasm dysfunction as primary complaint excluded	Secondary anorgasmia assessed via self-report, clinician rating and independent assessor rating	Sensate Focus (SF) weekly: testosterone, 10 mg/d (T) vs. diazepam, 10 mg/d (C) vs. SF monthly: T vs. diazepam (C); SF weekly: 16 sessions, SF monthly: 5 sessions	No difference in orgasmic ability between weekly vs. monthly T>diazepam frequency of orgasm; 6 mo follow-up (after drug discontinuation): gains maintained
Davis et al. (1995) [306]	Estradiol, Testosterone (n=16) Estradiol (n=17)	E&T: *M* age =57; E: *M* age =51; postmenopausal; 13/32 hysterectomy; 2/32 oophorectomy	Self-reported orgasmic dysfunction, assessed via Sabbatsberg self-rating scale	Estradiol (50 mg) plus testosterone (50 mg) (E&T) vs. estradiol (50 mg) (E), admininstered 3x monthly for 2 yr; checkups ever 6 mo	Both E&T and E tx increased orgasmic function; E&T > E greater orgasmic response

Note: SF=Sensate Focus

Table 8 . Pharmacological Treatments for Antidepressant-Induced Orgasmic Dysfunction

Reference	N	Subject Characteristics	Antidepressant	Definition of Anorgasmia	Treatment	Outcome
Placebo Controlled Outcome Studies						
Bupropion						
Masand et al. (2001) [312]	Bupropion (n=15) Placebo (n=15)	Impairment of sex drive, arousal and/or vaginal lubrication	N/A	Impairment in orgasm and orgasm satisfaction assessed via Arizona Sexual Experiences Scale	Bupropion SR (150 mg) daily for 3 wk vs. placebo daily for 3 wk	No change in bupropion tx in orgasm/ orgasm satisfaction; no difference bupropion vs. placebo
Buspirone						
Landen et al. (1999) [308]	Buspirone (n=16) Placebo (n=11)	MDD; decreased libido; orgasmic dysfunction (n=19)	Citalopram (min 40 mg/d) or paroxetine (min 30 mg/d)	Orgasm dysfunction assessed in interview via Udvalg for Kliniske Undersogelser scale	Buspirone (20 – 60 mg/d) for 4 wk vs. placebo; SSRI continued during tx	Change in orgasm function not specified
Buspirone, Amantadine						
Michelson et al. (2000) [307]	Buspirone (n=19) Amantadine (n=18) Placebo (n=20)	Depression; anxiety disorder; OCD; premenstrual syndrome; premenopausal or estrogen replacement; decreased arousal and pleasure	Fluoxetine dosage by group: B (31.4 mg/d), A (28.4 mg/d), and P (25.7 mg/d)	Impaired orgasm, assessed by clinician, self-report, daily diary, and Interview Rating of Sexual Function scale	Baseline and 4-wk dose, respectively: amantadine (50, 100 mg/d) buspirone (20,30 mg/d) vs. placebo; fluoxetine continued during tx	Improved orgasm in tx and placebo; no difference tx vs. placebo
Ephedrine						
Meston (2003) [311]	19	Female Sexual Arousal Disorder with complaints of decreased orgasm	Fluoxetine, sertraline, or paroxetine; min. 10 wk	Orgasmic ability, intensity/pleasure assessed via self-report	Two wk baseline, 8 wk crossover design placebo vs. 50 mg ephedrine 1 hr prior to sexual activity	Improved orgasm intensity/pleasure in ephedrine tx and placebo; no difference in orgasm tx vs. placebo

Table 8 . Pharmacological Treatments for Antidepressant-Induced Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Antidepressant	Definition of Anorgasmia	Treatment	Outcome
Ginkgo Biloba						
Kang et al. (2002) [310]	Ginkgo (n=4) Placebo (n=6)	Ginkgo group: *M* age= 47; placebo group: *M* age=46; depressive or anxiety disorders	Fluoxetine (20 mg/d), paroxetine (20-40 mg/d), or nortriptyline (30 mg/d)	DSM-IV diagnosis of sexual dysfunction; orgasm satisfaction and frequency via self-report and clinical interview	Ginkgo biloba at 120 mg/d for 2 wk, 160 mg/d for following 2 wk, 240 mg/d for final 4 wk vs. placebo doses on same schedule	No improvement in orgasm frequency or satisfaction in ginkgo vs. placebo; 8 wk: orgasm satisfaction improvement in placebo
Mirtazepine, Yohimbine, and Olanzapine						
Michelson et al. (2002) [309]	Mirtazepine (n=36) Yohimbine (n=35) Olanzapine (n=38) Placebo (n=39)	*M* age =36; depression; decreased vaginal lubrication	Fluoxetine (20 mg/d or greater)	Self-reported orgasmic inhibition, at least moderate in severity	Random assignment to mirtazapine (15-30 mg/d), yohimbine (5.4-10.8 mg/d), olanzapine (2.5-5 mg/d), or placebo, to be taken 1-2 h before sexual activity	No differences drug vs. placebo in diary or self-report ratings of orgasm function
No Control Outcome Studies						
Bupropion						
Walker et al. (1993) [313]	22	*M* age =45; depressed; libido decrease since fluoxetine tx	Fluoxetine (*M* dose= 25 mg/d)	Assessment of delayed or impaired orgasm via interview and self-report	2 wk washout followed by bupropion (75 mg b.i.d. – 150 mg t.i.d. daily) for 8 wk	84% complete and 10% partial resolution of orgasm dysfunction (results of female and male participants presented together)

Table 8 . Pharmacological Treatments for Antidepressant-Induced Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Antidepressant	Definition of Anorgasmia	Treatment	Outcome
Ashton & Rosen (1998) [314]	28	*M* age =42; affective or anxiety disorders; desire and arousal complaints	Paroxetine, fluoxetine, setraline, venlafaxine, fluvoxamine	Delayed orgasm or anorgasmia, assessed via clinical interview	Bupropion (75- 150 mg/d 1-2 h before sexual activity); if no response, 75 mg t.i.d./d for 3 days, 75 mg b.i.d./d for 3 days, and 75 mg t.i.d./d for 2 wk (longest tx period, 9 mo); SRI continued during tx	Improvement in 71% of orgasm complaints
Clayton et al. (2001) [315]	4	22-52 yo; MDD; decreased desire, arousal, and libido	Paroxetine (M dose=35 mg/d), sertraline (M dose=94 mg/d), fluoxetine (M dose=12.5 mg/d), and venlafaxine (M dose=225 mg/d)	Orgasmic ability via Changes in interview form of Sexual Functioning Questionnaire	Bupropion (150 m at start, 345 mg average ending dose) daily or twice daily, for 8 wk; SSRI discontinuation by 4 wk	4 wk: Improvement in orgasm subscale scores after discontinuation of SSRI
Gitlin et al. (2002) [316]	15	*M* age =41; History of MDD, dysthymic disorder, or depressive disorder NOS; not currently depressed	Fluoxetine (M dose=33 mg/d), sertraline (M dose=106 mg/d), paroxetine (M dose=31 mg/d), or citalopram (M dose=20 mg/d)	Self-reported orgasmic ability and satisfaction via Arizona Sexual Experiences Scale	Bupropion SR (100 – 150 mg) 2x daily for 7 wk	Improvement in ease of reaching orgasm, n.s. improvement in orgasm satisfaction

Table 8 . Pharmacological Treatments for Antidepressant-Induced Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Antidepressant	Definition of Anorgasmia	Treatment	Outcome
Ginkgo Biloba						
Cohen & Bartlik (1998) [317]	33	Decreased libido and orgasm dysfunction	Fluoxetine, nefazodone, bupropion, sertraline, paroxetine, venlafaxine, phenelzine, or vivactil	Delayed or inhibited orgasm through clinical interview or self-report	Ginkgo Biloba (40 – 60 mg) 2x daily up to 120 mg b.i.d./d for 4 wk; antidepressants continued during tx	Improvement in orgasmic functioning assessed via clinical interview and self-report
Granisetron, Sumatriptan						
Berk et al. (2000) [318]	16	*M* age =37; diagnoses include major depression, OCD, bulimia, social phobia, bipolar II disorder, panic disorder, trichotillomania, and/or borderline personality disorder	Clomipramine (125-250 mg/d), paroxetine (20-40 mg/d), sertraline (50-150 mg/d), fluvoxamine (100-300 mg/d), fluoxetine (20-40 mg/d), citalopram (20-60 mg/d)	Self-reported orgasm difficulty, frequency of difficulty, satisfaction, and intensity via Feiger scale	Sequential course of granisetron (1mg) and sumatriptan (100mg) 1 h before sexual activity	Improvement in orgasm difficulty and frequency of difficulty with granisetron; n.s. improvement with sumatriptan
Mianserin						
Aizenberg et al. (1999)[319]	16	*M* age =47; MDD, OCD, panic disorder, bipolar I disorder	Fluoxetine (20-40 mg/d), paroxetine (20-40 mg/d), fluvoxamine (200 mg/d), clomipradmine (75-150 mg/d)	Self-reported that orgasm function had "markedly decreased"	Mianserin (15 mg/d) at bedtime; SRI continued during tx	62% improvement in orgasm function: 7/16 restored normal orgasm function and 3/16 improved orgasm function to a minor disturbance

Table 8 . Pharmacological Treatments for Antidepressant-Induced Orgasmic Dysfunction (Ctd)

Reference	N	Subject Characteristics	Antidepressant	Definition of Anorgasmia	Treatment	Outcome
Mirtazepine						
Gelenberg et al. (2000) [320]	12	*M* age =43; improvement in MDD with SSRIs; 12/19 female in total sample	Fluoxetine (20 – 80 mg/d), sertraline (50 – 150 mg/d), paroxetine (20 mg/d)	Orgasmic dysfunction diagnosed by clinician via DSM-IV interview and self-reported through Arizona Sexual Experiences Scale	1 – 2 wk washout; Mirtazapine (7.5 mg h.s. - 45 mg/d) for 3-6 wk	6 wk: Improvement in orgasm function but not satisfaction (results of males and females reported together)
Sildenafil						
Nurnberg et al. (1999) [321]	7	*M* age =37; MDD, panic disorder, bipolar disorder; decreased libido and arousal; pain during intercourse	Fluoxetine (20-50 mg/d), sertraline (50-200 mg/d), nefazodone (150 mg/d), valproate (1 gm/d), trazodone (100 mg/d)	Anorgasmia or delayed/less intense orgasm, assessed by clinicians	Sildenafil (50-100 mg) 1-2 h before sexual activity	7/7 orgasm (less delay, more intense)
Salerian et al. (2000) [322]	31	Dissatisfaction with libido, arousal, orgasm and lubrication	64% of sample receiving SSRIs; other drug types include: TCAs, other antidepressants, benzodiazepines, mood stabilizers, stimulants, narcotics, antipsychotics	Satisfaction with orgasm assessed with Salerian Sexual Satisfaction Survey	Sildenafil (12.5 – 100 mg); duration of tx: 1 – 36 wk	Improved orgasm satisfaction; similar response regardless of psychotropic meds

Note: OCD=Obsessive Compulsive Disorder; MDD=Major Depressive Disorder; SRI=Serotonin Reuptake Inhibitor; SSRI= Selective Serotonin Reuptake Inhibitor; TCA=Tricyclic Antidepressants

self-directed masturbation training (bibliotherapy) and wait-list control, Heinrich [240] reported a 100% success rate for treating primary anorgasmia using therapist DM training at 2 month follow-up. Forty-seven percent of the bibliotherapy subjects reported becoming orgasmic during masturbation compared with 21% of wait-list controls. The effects of self-directed masturbation training were further investigated in a randomized trial comparing written versus videotaped masturbation assignments [241]. After 6 weeks, 65% of subjects using a text and 55% of women using videotapes had experienced orgasm during masturbation and 50% and 30%, respectively, were orgasmic during intercourse. None of the control women had attained orgasm. More recently, Hurlbert & Apt [242] compared the effectiveness of DM with coital alignment technique in 36 women with secondary anorgasmia. Coital alignment is a technique in which the woman assumes the supine position and the man positions himself up forward on the woman such that clitoral contact is maximized during coitus. Thirty-seven percent of woman receiving instructions on coital alignment technique versus 18% of those receiving DM reported substantial improvements (> 50% increase) in orgasmic ability during intercourse after only four 30-min sessions.

In summary, DM has been shown to be an empirically valid, efficacious treatment for women diagnosed with primary anorgasmia. For the woman with acquired anorgasmia who is averse to touching her genitals, DM may be beneficial. If, however, the woman is able to attain orgasm alone through masturbation but not with her partner, issues relating to communication, anxiety reduction, safety, trust, and ensuring the woman is receiving adequate stimulation either via direct manual stimulation or engaging in intercourse using positions designed to maximize clitoral stimulation (i.e., coital alignment technique) may prove more helpful.

b) Anxiety reduction techniques

Anxiety can serve as a distraction that disrupts the processing of erotic cues by causing the woman to focus instead on performance related concerns, embarrassment, and/or guilt. It can lead the woman to engage in self-monitoring during sexual activity, an experience Masters & Johnson [243] referred to as “spectatoring”. Some researchers have speculated that the increased sympathetic activation that accompanies an anxiety state may impair genital vasocongestion via inhibition of parasympathetic nervous system activity. Others have argued that SNS activation plays more of a facilitatory than inhibitory role in sexual arousal [244].

As originally conceived by Masters & Johnson [243], sensate focus involves a step-by-step sequence of body touching exercises, moving from non-sexual to increasingly sexual touching of one another's body. Components specific for treating anorgasmic women often include non-demand genital touching by the partner, female guidance of genital manual and penile stimulation and coital positions designed to maximize pleasurable stimulation. Sensate focus is primarily a couples' skills learning approach designed to increase communication and awareness of sexually sensitive areas between partners. Conceptually, however, the removal of goal-focused orgasm which can cause performance concerns, the hierarchical nature of the touching exercises, and the instruction not to advance to the next phase before feeling relaxed about the current one, suggest sensate focus is also largely an anxiety reduction technique and could be considered a modified form of in vivo desensitization.

The success of using anxiety reduction techniques for treating anorgasmia is difficult to assess because most studies have used some combination of anxiety reduction, sexual techniques training, sex education, communication training, bibliotherapy, and Kegel exercises, and have not systematically evaluated the independent contributions to treatment outcome. Of the controlled studies that have included anxiety reduction techniques, few have differentiated between treatment outcomes for lifelong versus acquired female orgasmic disorder. As can be seen in **Table 6**, across studies women have reported decreases in sexual anxiety and, occasionally, increases in frequency of sexual intercourse and sexual satisfaction with systematic desensitization, but substantial improvements in orgasmic ability have not been noted. Similarly, of the few controlled studies that have included sensate focus as a treatment component, none have reported notable increases in orgasmic ability. These findings suggest that, in most cases, anxiety does not appear to play a causal role in anorgasmia and anxiety reduction techniques are best suited for anorgasmic women only when sexual anxiety is coexistant.

c) Other Behavioral techniques

Ignorance about female anatomy and/or techniques for maximizing pleasurable sensations can certainly contribute to orgasm difficulties. Killmann and associates [245] compared the effectiveness of various

sequences of sex education and communication skills versus wait-list control on orgasmic ability in women with secondary anorgasmia. The authors found sex education to be beneficial for enhancing coital ability at post-test but not at 6-month follow-up. In a comparison study of the effectiveness of sex therapy versus communication skills training for secondary anorgasmia, Everaerd & Dekker [246] found both treatments were equally effective in improving orgasmic ability. As can be seen in **Table 6,** treatment comparison studies have generally found no differences in orgasmic ability between women whose therapy included using Kegel exercises versus those whose therapy didn't. To the extent that Kegel exercise may enhance arousal and/or help the woman become more aware and comfortable with her genitals, these exercises may enhance orgasm ability [239]. In summary, there is no direct empirical evidence to suggest that sex education, communication skills training, or Kegel exercises alone are effective for treating either primary or secondary anorgasmia. A review of studies suggests they may serve as beneficial adjuncts to therapy.

d) Pharmacological Approaches

There have been few placebo-controlled studies examining the effectiveness of pharmacological agents for treating Female Orgasmic Disorder. Of the few published, most examine the efficacy of agents for treating antidepressant-induced anorgasmia. Whether pharmacological agents would have the same treatment outcome effect on non-drug- versus drug-induced anorgasmia is not known.

• Non drug-induced anorgasmia

Using a single-blind design, Modell and associates [304] reported no significant effect beyond placebo of either 150 mg/day or 300 mg/day bupropion-SR on orgasm in 20 women with delayed or inhibited orgasm. Ito et al. [305] conducted a double-blind, placebo-controlled study of ArginMax, a nutritional supplement comprised of ginseng, Ginkgo biloba, Damiana leaf and various vitamins, on sexual function in 77 women with unspecified sexual function and reported a marginally significant group difference. It cannot be determined from the report how many women would meet a clinical diagnosis for anorgasmia. To date, there have been no published placebo-controlled studies on sildenafil for female anorgasmia **(Table 7).**

• Antidepressant-induced anorgasmia

As can be seen in Table 8, a number of case reports and open label studies report success in alleviating SSRI-induced anorgasmia with various agents. Findings from the few placebo-controlled studies published are less optimistic. Michelson et al. [307] examined the comparative effects of 8 weeks of treatment with either buspirone (20 mg/day ; n=19), amantadine (50 mg/day ; n=18), or placebo (n=20) on fluoxetine-induced sexual dysfunction in premenopausal women reporting either impaired orgasm or sexual arousal. The authors reported all groups experienced an improvement in orgasm during treatment, but neither busprione nor amantadine was more effective than placebo in restoring orgasmic function. At a higher dose level (mean daily dose = 47 mg), buspirone showed a marginally significant alleviation of sexual side effects in women taking either citalopram or paroxetine compared with placebo [308]. The authors did not distinguish between orgasm and desire disorders in either the classification of patients or treatment outcome. In a randomized, double-blind, parallel, placebo-controlled study of mirtazapine (15 mg/day), yohimbine (5.4 mg/day), olanzapine (25 mg/day) or placebo for fluoxetine-induced sexual dysfunction, Michelson et al. [309] found no significant improvement in orgasmic ability beyond placebo in 107 women with either impaired orgasm or vaginal lubrication. Kang et al. [310] reported no significant effect of Gingko-biloba beyond placebo in a small group of women with SSRI-induced sexual dysfunction. Meston [311] reported no significant effect of ephedrine (50 mg 1-hr prior to intercourse) beyond placebo on orgasmic function in 19 women with sexual side effects secondary to either fluoxetine, sertraline, or paroxetine treatment (Level 1 evidence).

In summary, to date there are no pharmacological agents proven to be beneficial beyond placebo in enhancing orgasmic function in women. Placebo-controlled research is needed to examine the effectiveness of agents with demonstrated success in case series or open-label trials (i.e., granisetron, and silenafil) on orgasmic function in women.

F. CONCLUSIONS AND RECOMMENDATIONS

We conclude that Directed Masturbation is an empirically valid and efficacious treatment for Lifelong Female Orgasmic Disorder (Grade A). To date, there are no empirically validated treatments for Acquired Female Orgasmic Disorder. Anxiety reduction techniques such as Sensate Focus and Systematic Desensitization have not been shown to be efficacious for treating either Lifelong or Acquired Female Orgasmic Disorder (Grade A). Anxiety reduction techniques may serve as beneficial adjuncts to therapy if the woman is experiencing a high level of anxiety (Grade B). There is no direct empirical evidence to suggest that sex education, communication skills training, or Kegel exercises alone are effective for treating either Lifelong or Acquired Female Orgasmic Disorder (Grade B). Of the few studies examining the effects of pharmacological agents for Female Orgasmic Disorder, none have been shown to be more effective than placebo (Grade A). Placebo-controlled research is essential to examine the effectiveness of agents with demonstrated success in case series or open-label trials (i.e., sildenafil, testosterone) on orgasmic function in women.

We recommend that future studies on women with orgasm difficulties conduct more careful classification of the disorder and better discriminate between women with Lifelong versus Acquired, and Generalized versus Situational Female Orgasmic Disorder. There is a paucity of research on the role of psychological, interpersonal, and social factors in the development of orgasm difficulties in women. Research is needed to examine the impact of learning and sexual scripts, relationship history, partner views, sexual experience, need to please partner, attitudes and beliefs toward sexuality and orgasm, and religious and cultural norms and expectations on orgasmic ability in women.

In order to better understand the physiology of women's orgasm, future studies are needed to :

1. **Further examine the differential brain activation during orgasm and during sexual arousal without orgasm.**
2. **Examine which specific serotonin receptor subtype(s) mediate the inhibitory effects of SSRI antidepressants on women's orgasm.**
3. **Assess whether prolactin secretion is a true specific indicator of orgasm in females and whether it serves as a biological "off switch" for sexual arousal in women.**
4. **Record uterine contractions during orgasm to better understand their role in women's orgasm and their relation with vaginal and rectal contractions.**
5. **Assess whether the vagus nerve is a real mediator of afferent supply from the human cervix/uterus.**
6. **Assess the physiological functions of the identified neuropeptides (e.g., VIP, Substance Y, CGRP, NO) in women's genitals.**

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